CLINICAL PROFILE OF COMPLICATIONS IN NEONATES BORN TO DIABETIC MOTHERS, BASED ON THEIR THIRD TRIMESTER GLYCEMIC CONTROL - A STUDY IN PATIENTS ADMITTED IN TIRUNELVELI MEDICAL COLLEGE HOSPITAL.

Dissertation submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the regulations

For the award of the degree of

M.D. (PEDIATRICS)

BRANCH – VII

TIRUNELVELI MEDICAL COLLEGE & HOSPITAL,

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY,

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APRIL 2013

CERTIFICATE

This is to certify that the dissertation entitled "**CLINICAL PROFILE OF COMPLICATIONS IN NEONATES BORN TO DIABETIC MOTHERS, BASED ON THEIR THIRD TRIMESTER GLYCEMIC CONTROL – A STUDY IN PATIENTS ADMITTED IN TIRUNELVELI MEDICAL COLLEGE HOSPITAL"** is the bonafide work of **Dr.R.Kavitha** in partial fulfilment of the requirements for the degree of **Doctor of Medicine in Paediatrics** Examination of The Tamilnadu Dr.M.G.R. Medical University to be held in April 2013.

DEAN

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DECLARATION

I, **DR.KAVITHA**, solemnly declare that dissertation titled, "**CLINICAL PROFILE OF COMPLICATIONS IN NEONATES BORN TO DIABETIC MOTHERS, BASED ON THEIR THIRD TRIMESTER GLYCEMIC CONTROL- A STUDY IN PATIENTS ADMITTED IN TIRUNELVELI MEDICAL COLLEGE HOSPITAL**" is a bonafide work done by me at the Department of Paediatrics, Tirunelveli Medical College Hospital during 2011 – 2012 under the guidance and supervision of **DR.T.KATHIR SUBRAMANIUM MD.,DCH** and **DR.S.DEVIKALA M.D.,D.C.H.,** Professors, TIRUNELVELLI MEDICAL COLLEGE, Tirunelveli. The Dissertation is submitted to **The Tamilnadu Dr.M.G.R. Medical University**, towards partial fulfilment of requirement for the award of **M.D. Degree (Branch – VII) in Paediatrics.**

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CONTENTS

ABBREVIATIONS

AGA	-	Appropriate for gestational age.
CNS	-	Central nervous system.
COA	-	Coarctation of aorta.
CBG	-	Capillary blood glucose.
DM	-	Diabetes mellitus.
5% D	-	5 % dextrose.
(eg)	-	Example.
Etc	-	Excreta.
ECG	-	Electrocardiography.
FBS	-	Fasting blood sugar.
FPF	-	Fibroblast pneumocyte factor.
FIG	-	Figure.
GDM	-	Gestational diabetes mellitus.
GIT	-	Gastrointestinal tract.
GCT	-	Glucose challenge test.
HbA_1C	-	Glycosylated haemoglobin.
HR	-	Hour.
HOCM	-	Hypertrophic cardiomyopathy.
HCT	-	Hematocrit.
IDDM	-	Insulin dependent diabetes mellitus.
IUD	-	Intrauterine death.

LGA	-	Large for gestational age.
LN	-	Labour naturalis.
LSCS	-	Lower segment caesarean section.
NICU	-	Neonatal intensive care unit.
OGTT	-	Oral glucose tolerance test.
PDA	-	Patent ductus arteriosus.
PCV	-	Packed cell volume.
PPBS	-	Post prandial blood sugar.
RDS	-	Respiratory distress syndrome.
RBS	-	Random blood sugar.
RBC	-	Red blood cell.
RL	-	Ringer lactate.
SGA	-	Small for gestational age.
TGA	-	Transposition of great arteries.
WKS	-	Weeks.

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INTRODUCTION

Diabetes mellitus during pregnancy is associated with adverse foetal and maternal outcomes. Improved health care and management of diabetes have reduced the incidence of adverse perinatal outcome in infants of diabetic mothers. Appropriate management of blood sugar in diabetic mother will maintain good glycemic control. In such cases, pregnancy outcomes could be expected comparable to the general population.

In general, abnormalities of carbohydrate metabolism occur frequently during pregnancy and about 3 to 5% of pregnant mothers will show glucose intolerance. Approximately 90% of these women will develop gestational diabetes.

Diabetes mellitus in pregnancy is classified into 2 types. They are

1. Pregestational diabetes.
2. Gestational diabetes.

Complications that occur in neonates born to diabetic mothers can be classified as those due to poor glycemic control in the 1st trimester (e.g.) congenital anomalies, and those due to poor glycemic control during the 3rd trimester (e.g.) metabolic complications. Even pre conceptional glucose control is essential to avoid congenital anomalies in neonates.

Management of diabetes in pregnant women includes watchful monitoring and treatment during antenatal period and treatment during delivery.

Tests done during the first trimester in diabetic mothers include:

- HbA_1C.
- Ultrasonography.
- Ophthalmic examination.
- Renal function test.
- Thyroid profile.

Tests done during second trimester in diabetic mothers include:

- Maternal serum testing for neural tube defects.
- Ultrasonographic survey including foetal echocardiography.
- In high risk cases - Chorionic villous sampling and amniocentesis.

Tests done during third trimester in diabetic mothers include:

- Monthly ultrasonographic examination for foetal growth monitoring.
- Weekly foetal surveillance using non stress test or biophysical profile.

To achieve good glycemic control, diabetic mothers are advised dietary modifications, exercise and medications. The objective of treatment is to maintain the fasting capillary glucose under 95mg/dl and

the 1hr and 2hr postprandial capillary glucose under 140mg/dl and 120mg/dl respectively. Normally Insulin therapy is advised to maintain glycemic control in pregnant women with diabetes. It has been demonstrated that human insulin analogues do not cross the placenta. Most recently, the oral hypoglycaemic agent Glyburide has been shown to be effective in the management of GDM, but it remains untested in pre gestational diabetes. Data are emerging that Metformin may also be used as an alternative to achieve glycemic control during pregnancy.

Glycosylated haemoglobin is a useful method to find out the glycemic control in the previous three months. In this study I have used this method to estimate the maternal third trimester glycemic control and compared it with the incidence of complications in babies born to them.

AIM AND OBJECTIVES

To study the pattern of complications in newborn born to diabetic mothers.

To assess the correlation between HbA_1C level in diabetic mothers and complications in babies born to them.

To emphasize the importance of strict glycemic control during pregnancy in diabetic mothers

To emphasize the need to monitor the babies born to diabetic mothers in their early neonatal period to recognise the complications early and treat them.

REVIEW OF LITERATURE

Diabetes mellitus is a disease of glucose intolerance which complicates 1% of the total pregnancies, making it the most common medical disorder that occurs during pregnancy. Diabetes mellitus in pregnancy presents as some peculiar complications in both mother and newborn. Planning in advance is important if one wants to have a baby without diabetes induced complications[1, 2]. Management of diabetes in pre gestational diabetes should aim at strict blood sugar control even before conception. Studies show that about 50% of gestational diabetic mothers develop Type - 2 Diabetes, later in their life[3].

Due to the recent advancements in the management of diabetes during pregnancy, there has been reduction in mortality and morbidity in neonates born to diabetic mothers. But perinatal mortality still continues at rates of 3 – 5 %, compared to about 1 – 2 % mortality noted in general population[2]. Incidences of complications such as neonatal hypoglycaemia, hyperbilirubinemia, respiratory distress syndrome, macrosomia, congenital malformation, polycythemia, hypocalcemia and birth injuries are still frequent in neonates born to diabetic mothers. These complications can be reduced by observing good glycemic control in diabetic mothers and the burden of perinatal mortality can be reduced.

Incidence

Incidence of diabetes mellitus is about 3 – 10 % of all pregnancies. Among that, gestational diabetes accounts for about 90% of cases of diabetes complicating pregnancy[2]. Mothers with gestational diabetes are individuals who have a genetic or metabolic predisposition towards diabetes and those who are not able to counter the diabetogenic changes that occur during pregnancy. Type - 2 diabetes accounts for about 8% of cases of diabetes mellitus in pregnancy.

Recent data shows that the prevalence of gestational diabetes has increased about 10 – 100 % [4]. A true increase in prevalence of gestational diabetes, aside from its adverse consequences in infants in the newborn period might also reflect to the pattern of increased diabetes and obesity especially in the babies. Therefore, meticulous screening of pregnant mothers for gestational diabetes and management of diabetic mothers in order to achieve a good glycemic control are mandatory for the healthy society in future. Maternal complications that occur during and after pregnancy could also be reduced.

Physiology

Pregnancy by itself is a diabetogenic state. The reason for this is the hormonal changes that occur during pregnancy. Most important of these is the increase in insulin resistance. This is mainly attributed to the increase in the placental hormone, the human placental lactogen that is

found in increased amounts during the third trimester of the pregnancy. This explains why gestational diabetes occurs more after 26 weeks[2, 4].

Changes in carbohydrate metabolism during pregnancy:

As a result of many hormonal changes that occur during pregnancy, the carbohydrate metabolism in pregnancy undergoes many changes. The most important reason why pregnancy increases the diabetogenic tendency of asymptomatic mother is the progressive increase in insulin resistance that occurs during pregnancy. Other reasons for this increased diabetogenic tendency are the increased lipolysis and the changes in gluconeogenesis which usually occurs during pregnancy[2, 4, 5]. During early pregnancy, there is increased sensitivity to insulin and the patients tend to have an increased incidence of hypoglycaemia. This increased insulin sensitivity is due to high levels of estrogen. Fasting blood sugar is usually lower than normal from 10^{th} to 16^{th} week of pregnancy, and then the blood sugar values rise gradually up to 32 weeks. During the 3^{rd} trimester, there is an increase in insulin resistance which leads to increased blood sugar values. Increased level of human placental lactogen hormone is the main reason for the insulin resistance that occurs during the second half of pregnancy[5]. Other causes are increased insulin catabolism by placental insulinases and the relative dysfunction of insulin by various hormones that increase during pregnancy. This is the reason for the increased incidence of gestational diabetes after 26 wks of

pregnancy and the need to screen for gestational diabetes during that period is emphasized[12].

Table.1

Diabetogenic Hormones in Pregnancy [13]

Hormone	Peak elevation (wks)	Diabetogenic potency
Prolactin	10	Weak
Estradiol	26	Very weak
Cortisol	26	Very strong
Progesterone	32	Strong

Effects of Diabetes on Pregnancy:

Maternal:

Mostly carbohydrate imbalance is asymptomatic in pregnancy. In some diabetic mothers with microalbuminuria, worsening albuminuria during pregnancy is at increased risk for preeclampsia (About 10 – 25 % increased risk) [2, 14, 15].

Infection - Incidence of chorioamnionitis and postpartum endometritis is increased in pregnancies complicated by diabetes.

Diabetic gastroparesis – This exacerbates nausea and vomiting that is usually present in normal pregnancies which in turn lead to the need for extra nutritional support for diabetic mothers[2].

Diabetic retinopathy may develop or retinopathy that is already present may worsen during pregnancy complicated by diabetes. Patients who have pre-existing renal disease are at high risk for further decline in renal function during pregnancy. Post partum bleeding is high due to exaggerated uterine distension.

Hyperglycemia / Hypoglycemia and Ketoacidosis are common. Coronary artery disease and thromboembolic complications may occur.

Major birth defects in infant of diabetic mother:

The incidence of foetal malformations is 4-10 times more in individuals with uncontrolled diabetes at the time of conception and normal blood glucose during the preconception period and during the entire period of organ development in foetus should be maintained[6, 8].

CNS-SKELETAL	-	Neural tube defects, Sacral agenesis, Holoprocencephaly.
CARDIOVASCULAR	-	HOCM, TGA, COA, PDA.
RENAL	-	Renal agenesis, Hydronephrosis.
GIT	-	Duodenal atresia, Lazy left colon syndrome.

Metabolic Complications:

Pederson's theory of HYPERGLYCEMIC – HYPERINSULINISM [7] is quoted to explain the metabolic complications in the newborn born to diabetic mothers. According to this, uncontrolled maternal hyperglycaemia causes foetal hyperglycaemia. This in turn stimulates foetal islet cells to produce hyperinsulinemia. This foetal hyperinsulinemia is said to be the primary cause for macrosomia, respiratory distress syndrome and other metabolic complications.

Hypoglycemia

Hypoglycemia is seen in 25 – 40 % of babies born to diabetic mothers[2, 15, 21]. Neonatal hypoglycemia is the most frequently presenting complication among those seen in infants born to diabetic mothers. According to the recent guidelines, blood glucose value of < 40mg/dl is hypoglycaemia. It is mainly due to excessive insulin production by pancreatic islet cells of the newborn, which are enlarged and hyperactive due to maternal hyperglycaemia which causes foetal hyperglycaemia and stimulates the islet cells. Meticulous regulation of blood sugars, especially during the second and third trimester of pregnancy may reduce these complications. Mostly it is asymptomatic, but the various presenting features are

- Jitteriness
- Seizure
- Lethargy
- Refusal of feeds
- Respiratory distress
- Cyanosis
- Shock
- Incessant cry etc.

These babies should be admitted in neonatal intensive care unit. Periodic blood glucose monitoring is a must. In asymptomatic cases, frequent breast feeding is advised. In symptomatic cases, IV 10% Dextrose 2ml/kg should be given as a bolus. This should be followed up with glucose infusion starting from a glucose infusion rate of 4 to 6 mg/kg/min. This is to prevent rebound hypoglycemia. Hypoglycemia which persists even with a glucose infusion rate of 12 mg/kg/min is called resistant hypoglycemia. In such cases drugs such as Glucagon, Hydrocortisone and Diazoxide are used [16, 17].

Hypocalcemia

Hypocalcemia is seen in 25 – 30% of babies born to diabetic mothers.[21] Mostly it is asymptomatic. Usually it presents during first three days of life. Contributory factors may be prematurity, birth asphyxia

and inappropriate response of Parathormone to hypocalcemia. It may present as

- Jitteriness
- Apnoeic attacks
- Hypotonia
- Seizures.

Ionised calcium value < 4 mg/dl is required to label as hypocalcemia. But S. Calcium (total) value < 7mg/dl is also taken as hypocalcemia[16,18,20]. Hypocalcemia in well babies born to diabetic mothers, usually resolves without any treatment and we do not routinely measure serum calcium in asymptomatic infants. Hypomagnesemia may coexist[16,17].

Hypomagnesemia should be considered in infants with hypocalcemia, because hypocalcemia does not respond until the hypomagnesemia is corrected. Babies found to have hypocalcemia are admitted in NICU and Inj.10% Calcium gluconate is given as a slow intravenous injection and then maintained with oral calcium of 75 -100 mg/kg/day.

Hyperbilirubinemia

Hyperbilirubinemia occurs in 20 – 25 % of infants born to diabetic mothers[2,16,17,21]. It is seen with increased frequency in infants born to diabetic mothers because of the following reasons,

- Bilirubin production is accelerated.
- Insulin causes increased stimulation of erythropoietin.
- The life span of red blood cells is reduced because of less deformable cell membranes due to glycosylation of erythrocyte cell membrane. This leads to destruction of red cells as they traverse through the micro circulation.
- Other factors that may be a cause for jaundice are prematurity, defective hepatic conjugation of bilirubin and an increased enterohepatic circulation of bilirubin due to poor feeding.
- Infants born to diabetic mothers with good glycemic control, have fewer problems with hyperbilirubinemia. The increasing gestational age of infants at delivery leads to decreased incidence of hyperbilirubinemia. Hyperbilirubinemia in infants born to diabetic mothers is diagnosed and treated as in any other infant. Treatment is individualised in each neonate and depends on the value of serum bilirubin, the infant's gestational age, weight of the neonate, clinical status and day of life. Either phototherapy or exchange transfusion is done according to the need.

Polycythemia

Polycythemia is seen in 30 - 35 % of infants born to diabetic mothers. Chronic foetal hypoxia leads to polycythemia that occurs in

infants born to diabetic mothers[16,17,19,21]. Polycythemia is defined as venous hematocrit of over 65%.

A venous hematocrit of over 64% or more at 2 hrs of age.

An umbilical venous or arterial hematocrit of over 63% or more.

The mean venous hematocrit of term infants is 53% in cord blood, 60% at 2 hours of age, 57% at 6 hours of age, and 52% at 12 to 18 hours of age.

Symptoms

- Poor feeding.
- Lethargy.
- Apnoea.
- Seizures.
- Cyanosis.
- Respiratory distress.
- Increased jaundice.

Hematocrit > 65% can result in intravascular thrombosis.[19] Thrombosis has been described in the arterial system, including the aorta but is particularly prone to occur in the veins. Polycythemia leads to increased incidence of hyperbilirubinemia due to the increased red blood cell destruction during a particular period of time. Treatment is PARTIAL EXCHANGE TRANSFUSION. Indicated in asymptomatic babies with PCV above 70% and in symptomatic babies with PCV above

65%.It is done by exchanging blood of the baby with normal saline. The amount to be exchanged is calculated using the formula

VOLUME OF EXCHANGE IN ML = (OBSERVED HCT-DESIRED HCT)/OBSERVED HCT × BODY WT × BLOOD VOLUME PER KG.

MACROSOMIA

Foetal macrosomia is defined as foetal weight equal to or larger than 4kg. Macrosomia may be the reason for the increased incidence of caesarean section or obstetric trauma such as Fracture clavicle, Erb's palsy and Klumpkey's palsy as a result of shoulder dystocia[2,16,17.] Macrosomia is found to be associated with elevated third trimester maternal blood sugar, hyperinsulinemia and hypoglycaemia. Hence all pregnant diabetic mothers should undergo ultrasound examinations every 4 weeks, starting at 20 weeks of pregnancy to estimate the foetal weight and to follow the foetal growth. The first indication of developing macrosomia is an increase in abdominal circumference than other measurements, resulting in elevated head to abdomen and femur to abdomen ratios. The estimated foetal weight may be in the 60^{th} to 80^{th} percentile between 26 and 32 weeks but by the end of the pregnancy will be above the 90^{th} percentile. Primary management of macrosomia includes caesarean section if the foetal weight exceeds 4 kg. All macrosomic babies should be admitted and kept under observation to look for other complications such as hypoglycemia.

Respiratory Distress Syndrome

Respiratory distress syndrome is seen in 3 – 5 % of babies born to diabetic mothers[21]. One of the causes of respiratory distress in infant of diabetic mothers is hyaline membrane disease. Insulin antagonises the stimulatory effects of cortisol on fibroblasts to induce the synthesis of fibroblast pneumocyte factor (FPF) which in turn inhibits type-1 cells and phosphatidyl choline production. Measurement of phosphatidyl glycerol alone or in combination with the lecithin, phosphatidyl choline may be a more reliable indicator of lung maturity in diabetic pregnancies than the lecithin - sphingomyelin ratio alone[16,20]. Besides RDS, there are many other causes of respiratory distress in infants born to diabetic mothers. These include

Causes of respiratory distress in infants born to diabetic mothers

- Transient tachypnea of the newborn.
- Polycythemia.
- Birth asphyxia.
- Hypertrophic cardiomyopathy.
- Other cardiac or pulmonary anomalies.
- Pneumonia.
- Pneumothorax.
- Diaphragmatic hernia.

Effects of pregnancy on diabetes[14,15]:

- More insulin is necessary to achieve metabolic control.
- Progression of diabetic retinopathy.
- Worsening of diabetic nephropathy.
- Increased risk of death for patients with diabetic cardiomyopathy.

These patients have a tendency toward metabolic instability and will need frequent blood glucose monitoring, continuous adjustments in therapy, and a highly regulated lifestyle. For diabetic patients who already have organ damage, pregnancy may accelerate end organ disease, requiring intensive testing and therapeutic procedures. The complex interaction between abnormal carbohydrate metabolism and pregnancy should be clearly explained to each patient immediately after the diagnosis is made and prior to pregnancy when the patient has pre gestational diabetes.

Classification of diabetes mellitus in pregnancy

Pre gestational

- Type-1
- Type-2

Gestational[22]

- Diet controlled
- Insulin requiring

Type-1

Diabetes is a condition in which body makes no insulin or so little insulin that the body cannot change blood sugar into energy. This occurs during childhood or adolescence[20, 22].

Type-2

Diabetes is a condition in which body makes too little insulin or cannot use the insulin it makes to change blood sugar into energy. This occurs at child bearing age[20, 22].

GDM- Diabetes first diagnosed in a pregnant woman usually resolves after pregnancy.

Whites classification

Named after PRISCILLA WHITE[2], who did research on the effect of diabetes mellitus on perinatal outcome is widely used to assess maternal and foetal risk.

CLASS A1 - GDM, Diet controlled.

CLASS A2 - GDM, Medication controlled.

CLASS B - Onset at age > 20 yrs /Duration <10yrs.

CLASS C - Onset at 10 - 19 yrs / Duration 10-19 yrs.

CLASS D - Onset before 10yrs / Duration >20yrs.

CLASS E - Overt diabetes with calcified pelvic vessels.

CLASS F - Diabetic nephropathy.

CLASS R - Diabetic retinopathy.

CLASS RF - Retinopathy with nephropathy.

CLASS H - Ischaemic heart disease.

CLASS T - Prior kidney transplant.

Table. 2

Complications in pregnancy by type of diabetes.[23]

Complications	Normal	GDM	Type-1 Dm	Type-2 Dm
Con. Anomalies	1-2.2	1-2.2	10	10-15
Miscarriages	5-15	-	9-17	8-15
Still birth	0.5	0.5	2.5	1-1.5
Preterm	4-11	4-11	22-25	26-46
Macrosomia	8-10	17-29	9-28	9-14
Hypoglycemia	-	1	5-25	51
RDS	-	-	2-6	40
NICU Care	-	29	-	37-40

Risk factors for GDM

- Age > 30 yrs.
- Obesity [22].
- History of GDM during previous pregnancy.
- History of LGA baby during previous pregnancy.
- Family history of type - 2 DM.

- Primigravida [2,22,25].
- Higher dietary fat with low carbohydrate intake during pregnancy.
- Eating one or more egg / day before or during pregnancy increases the risk of GDM.[24]

Screening for GDM

- Done in high risk pregnancies.
- Done by using glucose challenge test and glucose tolerance test.

Glucose Challenge Test

By using 50gms glucose challenge test without regard to time of day/last meal between 24-28wks of pregnancy. A plasma glucose value of 140mg% or that of whole blood of 130mg% at 1hr is considered as a cut off point for consideration of a 75gm glucose tolerance test.

The diagnosis of GDM is based on a 75gm GTT. A larger than usual glucose load (75gms) is advocated as there is an increased turnover of glucose in pregnant state[2,26].

Indications for GTT

- Fasting glycosuria on one occasion before 20th wk and 2 or more occasions thereafter.
- Following a positive screening test.
- If RBS is >95mg/100ml.
- If FBS is >126mg/dl and if it is confirmed on repeat test, there is no need to perform GTT.

Glucose Tolerance Test

Method Used

The patient is advised to come with fasting for about 8 hrs.

Before starting the procedure a blood sample is collected which gives the fasting blood sugar. Then the patient is asked to take about 75 gm sugar, and 3 more blood samples are taken after 1hr, 2hr and 3hr respectively.

Interpretation of Results:

- Normal values - Fasting (60-100 mg/dl).
- 1hr value- <200 mg/dl.
- 2hr value - < 140 mg/dl.

Table.3

Diagnostic Criteria For GDM[28]

	100 gm glucose	**75 gm glucose**
Fasting	95 mg/dl.	95 mg/dl.
1hr	180 mg/dl.	180 mg/dl.
2hr	155 mg/dl.	155 mg/dl.
3hr	140 mg /dl.	-

Diagnosis is based on more than two values listed above.

Diagnostic Criteria for DM Prior To Pregnancy

- Symptoms of DM + RBS > 200mg/dl.
- FBS > 126mg/dl.
- 2hr PPBS > 200mg/dl during OGTT. (With 75gm glucose in water).

Glycosylated Haemoglobin:

HbA1C is a blood test to determine the level of haemoglobin that is sugar coated. As the lifespan of RBC is 120 days, HbA_1C values reflect the adequacy of glucose control for the previous 4-6wks. HbA_1C level >7 indicate elevated glucose during the past 4-6wks and is associated with increased incidence of complications.[29] Recent guidelines insist on maintenance of HbA_1C level < 6.[30]

Methods For Measuring HbA_1C

1. Methods based on structure differences
    a) Immunoassays.
    b) Affinity chromatography.
2. Methods based on charge differences.
    a) Ion exchange chromatography.
    b) Electrophoresis.
    c) Isoelectric focussing.
    d) High pressure liquid chromatography.

Limitation of HBA_1C Measurement

Although HbA_1C levels are a reliable indicator of recent average glycemic control, they do not provide information about the daily pattern of blood glucose fluctuation which is required for fine tuning of insulin doses.[32]

Guidelines for treatment in DM complicating pregnancy

Prepregnancy-

Explain general risks and management of diabetes in pregnancy. Evaluate any additional risks[2,14,20].

- Evaluation of blood pressure.
- Evaluation of renal status.
- Evaluation of retinal status.
- ECG.
- Clinical evaluation of peripheral and autonomic neuropathy.
- Clinical evaluation of hypoglycemic symptoms.
- Clinical evaluation of peripheral vascular disease.

Discuss effective contraception until good glucose control.

Contraception methods[1]

Method of choice - Barrier method.

Low dose oral contraceptive pills may be safely used.

Folate supplementation (4-5mg) daily for at least 2 months before and during first trimester[2, 16].

Antenatal [33]

- Screen for GDM ideally in all pregnancies.
- Regular capillary glucose series.
- Avoid oral hypoglycemic agents.
- Appropriate diet.
- Follow insulin regimen to keep capillary glucose levels as normal as possible.
- Regular ophthalmological review.
- Monitor for hypertensive disease.
- Foetal surveillance.

Tests of Fetal Well Being [34:]

- Counting of foetal movements done every night from 28wk. Ten movements in < 60min is reassuring.
- Non stress test - Done twice weekly. Begin at 28-34 wks in patients with IDDM. Two heart rate accelerations in 20 min is reassuring.
- Contraction stress test is done weekly. No heart rate deceleration in response to < 3 contractions in 10 min.
- Ultrasound biophysical profile, done weekly.

Labour and Delivery

Timing can be delayed until term if blood sugar is controlled.

Maintain good perinatal glucose control.

Mode of delivery depends on maternal and foetal complications.

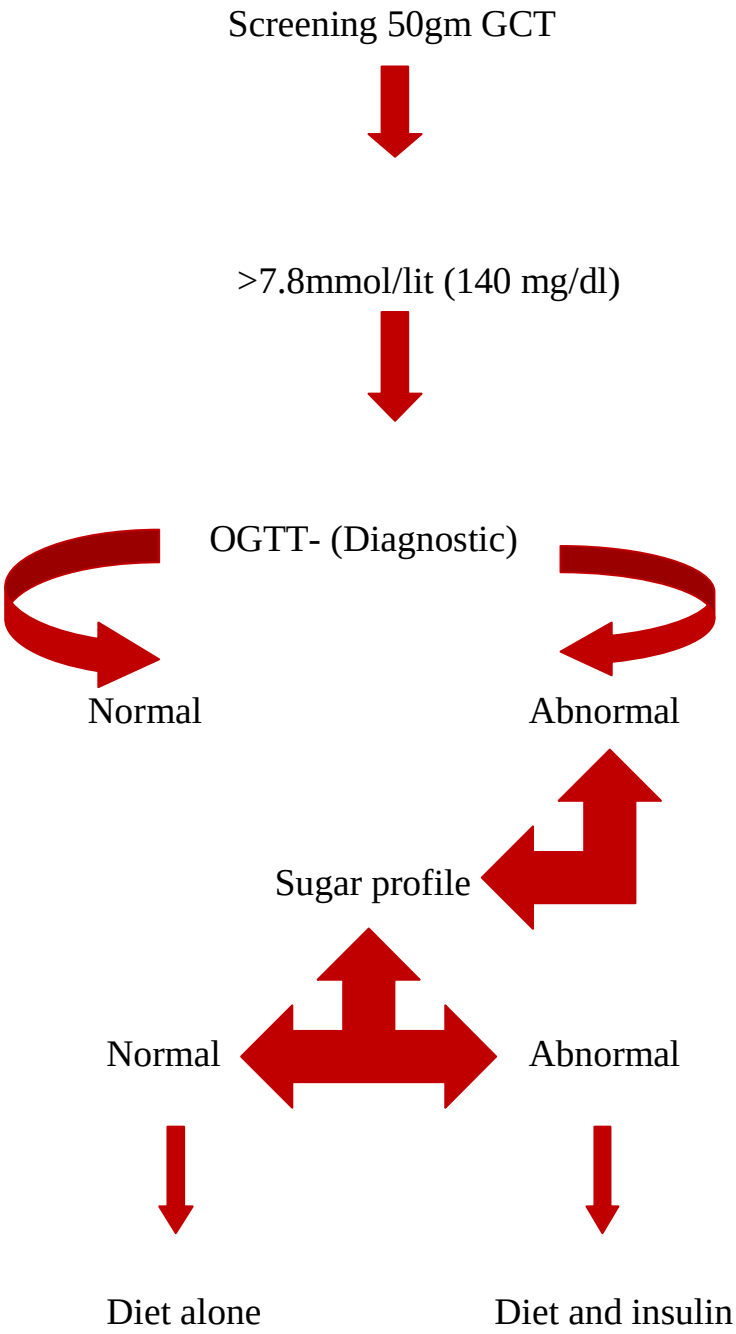
Postnatal

- Insulin requirement decreases during postnatal period.
- Continue CBG monitoring.
- Encourage breast feeding.

Benefits of Breast Feeding:

- Improved glucose metabolism.
- Non insulin mediated use of glucose by mammary gland to synthesize lactose.[35]
- Increased insulin sensitivity due to increased prolactin and decreased oestradiol.
- Improved B cell function.
- Improved lipid metabolism.

MANAGEMENT PROTOCOL FOR GDM



(Monitor weekly sugar profile)

Management for DM complicating pregnancy:

Antenatal:

Control of blood sugar remains the mainstay of management during the antenatal period.

Methods used

DIETARY MANAGEMENT - A meal plan to maintain euglycemia should be followed[2, 36]. Total calories 30-35 kcal/kg given as 3 meals and 3 snacks daily. Diet should contain 55% carbohydrate, 20% protein, 25% fat (with <10% saturated fat).

GLYBURIDE- A sulfonylurea which acts by closing potassium channels of the pancreatic beta cells causing calcium influx and secretion of insulin from storage granules[37]. It also decreases insulin resistance. Dose - 1.25 or 2.5 mg twice daily, max. dose upto10mg twice daily.

INSULIN THERAPY- If PPBS is >150 mg/dl, in spite of dietary control, plain insulin is given subcutaneously in 3 divided doses started. Amount of insulin depends on blood sugar levels.

Blood sugar should be controlled to maintain fasting blood sugar <95 mg/dl and postprandial <140 mg at 1hr, <120 mg at 2hr.

Intrapartum

Glucose infusion is provided to all patients in labour as 5%D in RL. (Rate -125 ml/hr)

Bedside glucose monitor- Monitor every 2-4hrs in early labour and 1-2hrs in active labour. For patients on insulin, continuous insulin infusion of regular insulin 25U in 250ml of saline (0.5-2.0U/hr). Continuous foetal monitoring is mandatory.[38]

Postpartum

Immediately after delivery of the placenta, there is a sudden loss of insulin resistance and the majority of patients will not require insulin for 24-48 hrs. Once their fasting/postprandial capillary glucose starts to rise, insulin therapy should be restarted using one half to two thirds of the dosage that the patient was receiving before delivery. This initial dose is adjusted according to the patient's response.

METHODOLOGY

TITLE OF THE SYUDY:

Clinical profile of complications in newborn born to diabetic mothers based on their third trimester glycemic control – A study in patients admitted in Tirunelveli Medical College Hospital.

AIM OF THE STUDY:

- To study the pattern of complications in newborn born to diabetic mothers.
- To assess the correlation between HbA_1C level in diabetic mothers and complications in babies born to them.

Type of Study

Cohort study.

Study Period

The study was conducted for a period of 12 months (from Aug 2011 to July 2012).

Settings

Babies born to diabetic mothers were examined clinically and by biochemical investigations to find out any complications due to maternal diabetes. Retrospectively, mothers third trimester glycemic control was assessed by perusal of previous history and laboratory investigation records and also by doing HbA_1C levels during postnatal period. Correlation between the incidence of complications

in neonates born to diabetic mothers was assessed with third trimester glycemic control of the mother. As $HbA_1C < 6$ is considered as good glycemic control in recent guidelines, mothers in this study were classified as with $HbA_1C < 6$ and with $HbA_1C > 6$ for better correlation.

Sample Size:

52 babies born to diabetic mothers who came under the inclusion criteria were included in the study.

Inclusion Criteria:

1) Neonates born to gestational diabetic mothers.
2) Neonates born to mothers with pre gestational diabetes.

Exclusion Criteria:

Complications in neonates born to diabetic mothers which reflect first trimester glycemic control (e.g.) congenital malformation.

Tools used:

Investigations done in babies :

- Blood sugar levels at 1hr, 2hr, 3hr, 6hr, 12hr, 24hr, 36hr and 48hr.
- Serum bilirubin (Total, Direct and Indirect).
- PCV at 1hr and 24hrs.
- Serum calcium.
- Laboratory investigations in mother: HbA_1C was done in mothers as it gives the last trimester glycemic control.

Data Analysis:

Data collected were entered in Excel spread sheet and analysed using SSPS version 16. Simple calculations like percentages, proportions and mean values were derived. Appropriate statistical tests like chi-square test, T test were used.

OBSERVATION

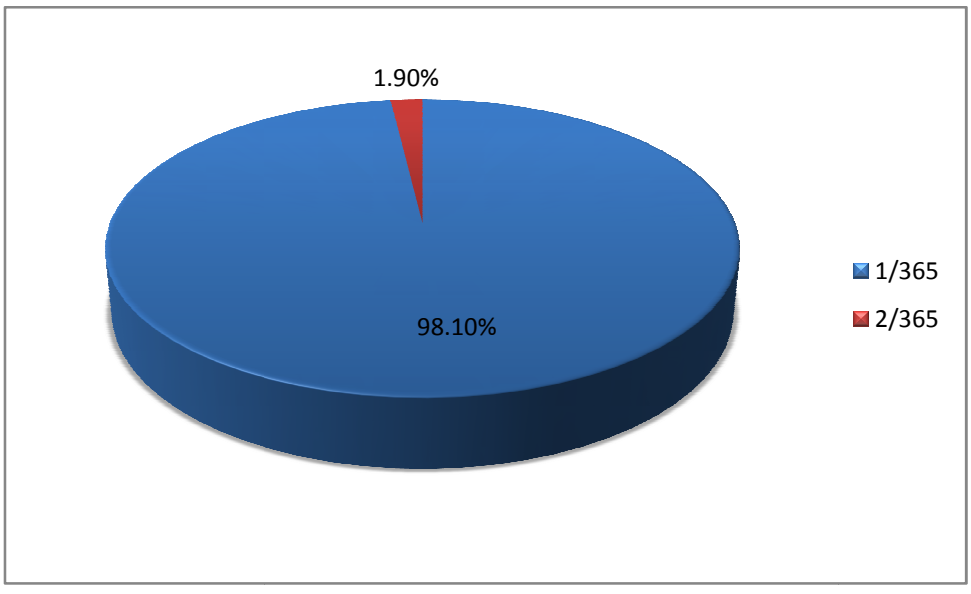
In this study, 52 cases were studied. Among those 52 cases, 51 cases (98.1%) were included in my study in their first day of life as most of the babies included in this study were born in the same hospital where study was conducted and 1 case (1.9%) was included on second day of life.

Table.4

AGE

Age	Frequency	Percentage
1/365	51	98.1%
2/365	1	1.9%

Fig.1



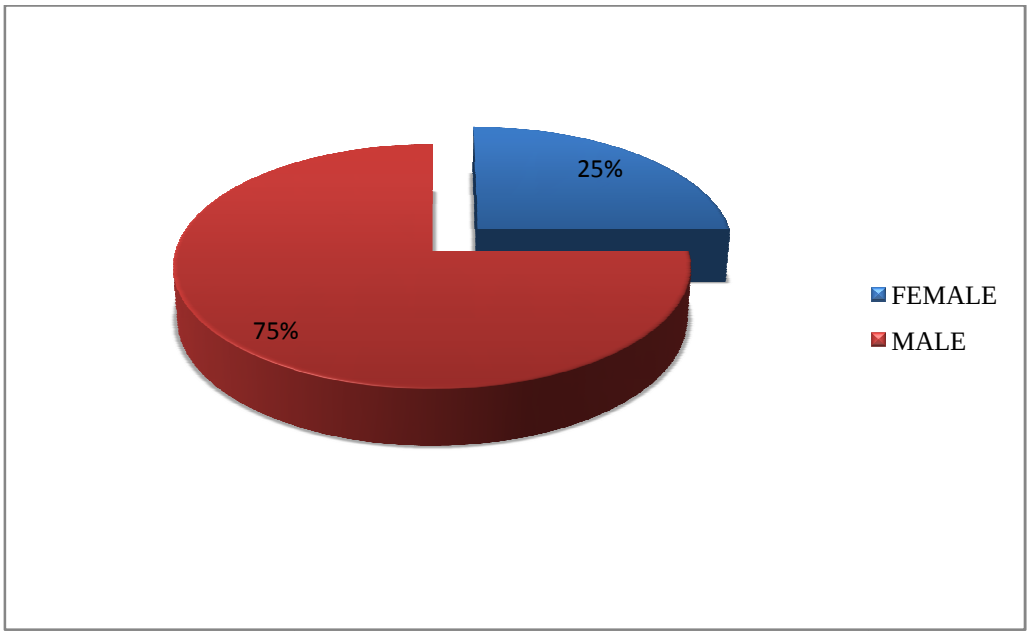
SEX

Out of the 52 cases studied 13 cases (25%) were females and 39 cases (75%) were males.

Table. 5

Sex	Frequency	Percentage
Female	13	25.0%
Male	39	75.0%

Fig.2



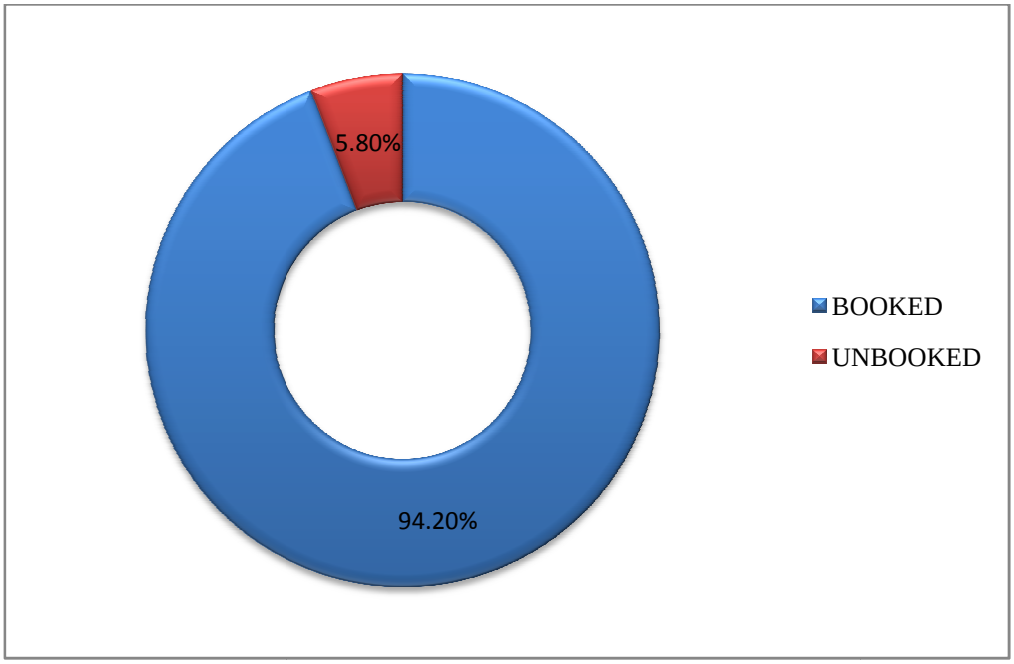
BOOKED/UNBOOKED:

Among the 52 cases, 3 cases (5.8%) were booked cases and 49 cases (94.2%) were unbooked.

Table.6

Booked / Unbooked	Frequency	Percentage
Booked	3	5.8%
Unbooked	49	94.2%

Fig. 3



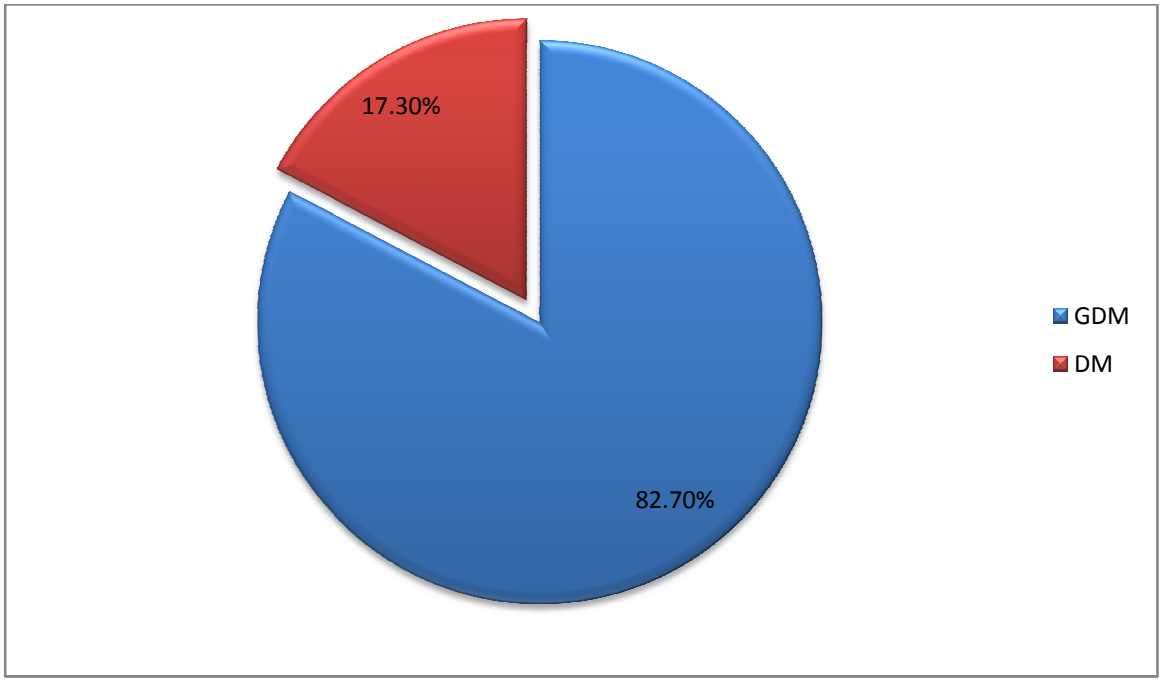
GDM/DM:

Among the mothers studied, 9 mothers (17.3) were having pregestational diabetes and 43 mothers (82.7%) were having gestational diabetes.

Table – 7

GDM / DM	Frequency	Percentage
DM	9	17.3%
GDM	43	82.7%

Fig.4



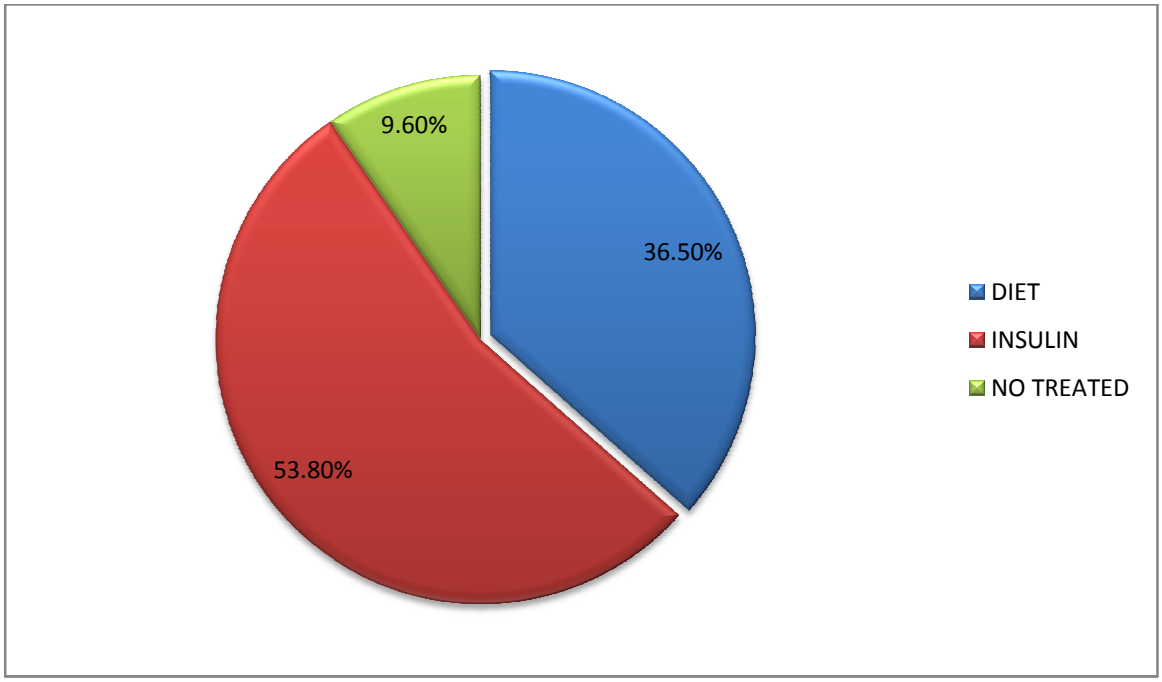
TREATMENT DETAILS:

Out of the 52 cases, 47 cases (90.3 %) were treated and 5 cases (9.6%) were not treated. Among the treated cases, 19 cases (36.5%) were on diabetic diet and 28 cases (53.8%) were on insulin.

Table.8

Treatment	Frequency	Percentage
Diet	19	36.5%
Insulin	28	53.8%

Fig.5



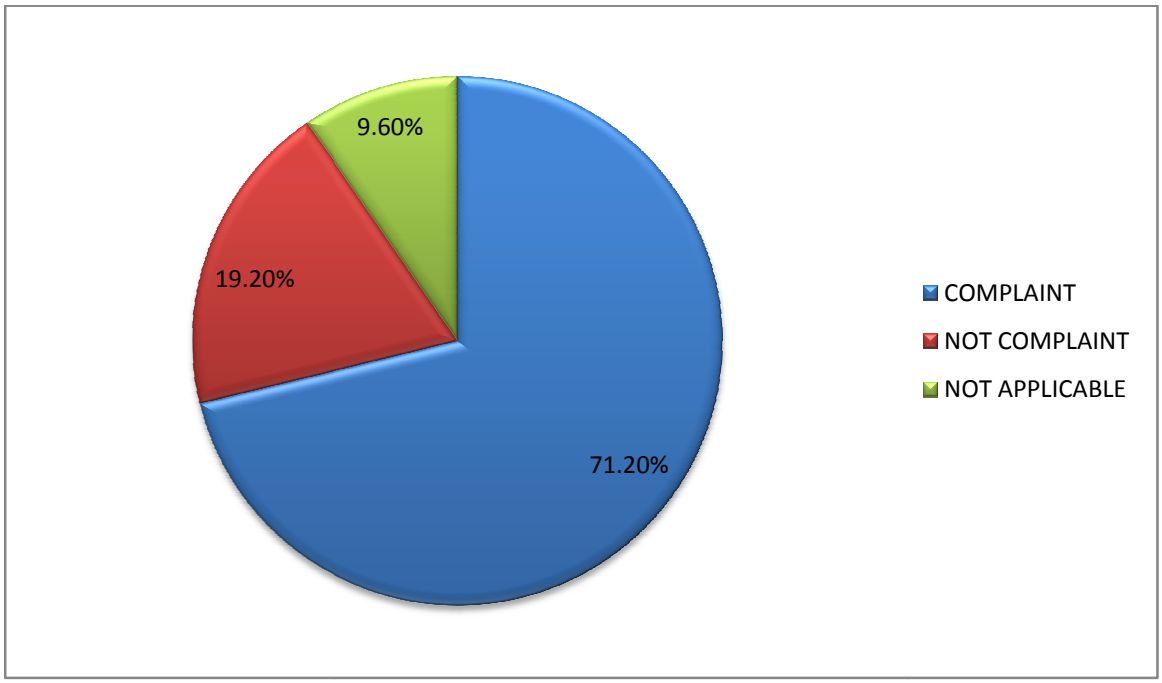
COMPLIANCE:

Among the 47 treated cases, 37 cases were compliant and 10 cases were not compliant. Those who were not treated were included as not applicable.

Table.9

Compliance	Frequency	Percentage
COMPLIANT	37	71.2%
NOT COMPLIANT	10	19.2%
NOT APPLICABLE	5	9.6%

(FIG – 6)



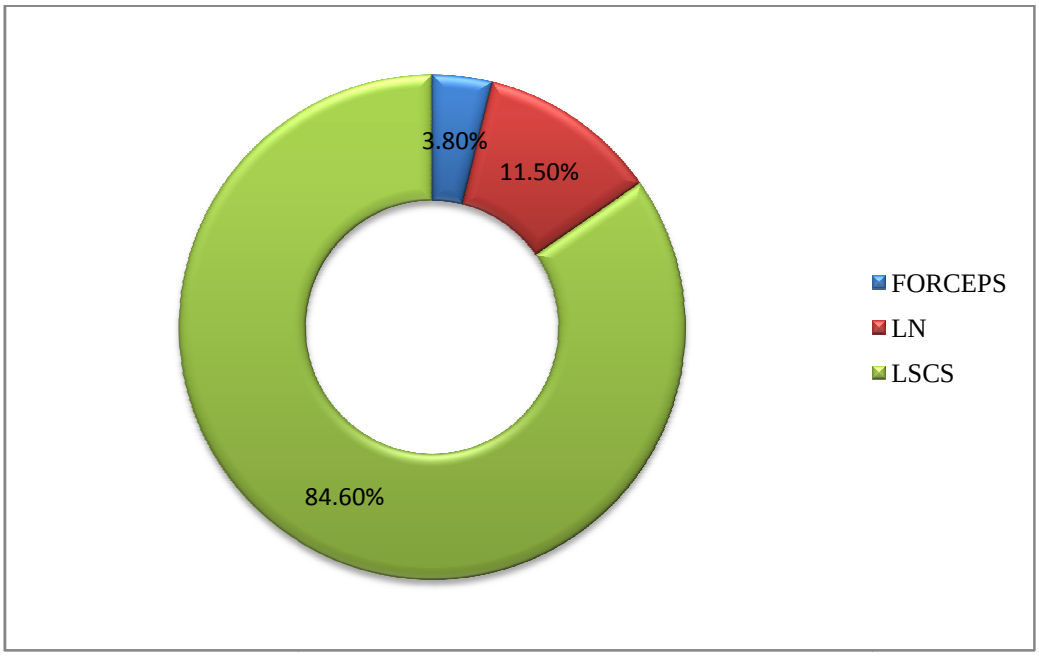
MODE OF DELIVERY:

Among the 52 babies, 2 of them were delivered by forceps delivery, 6 of them were delivered by labour naturalis and 44 of them were delivered by LSCS.

Table – 10

Mode of delivery	Frequency	Percentage
FORCEPS	2	3.8%
LN	6	11.5%
LSCS	44	84.6%

FIG – 7



ASPHYXIA:

Out of the 52 cases,

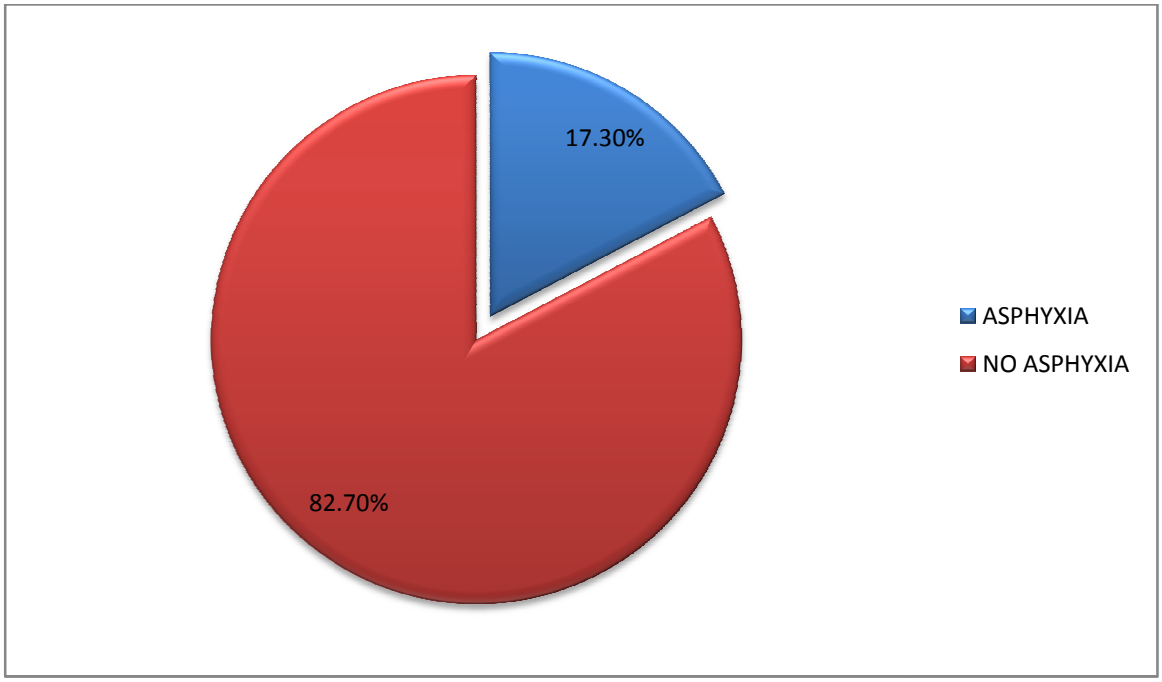
- 9 cases (17.3 %) were asphyxiated.

- 43 cases (82.7 %) were not asphyxiated.

TABLE – 11

ASPHYXIA	FREQUENCY	PERCENTAGE
YES	9	17.3%
NO	43	82.7%

FIG – 8



BIRTH INJURIES:

Out of the total no of cases,

6 cases had birth injuries. Out of them, 3 babies were delivered by normal labour, 2 babies were delivered by forceps delivery and 1 baby was delivered by caesarean section.

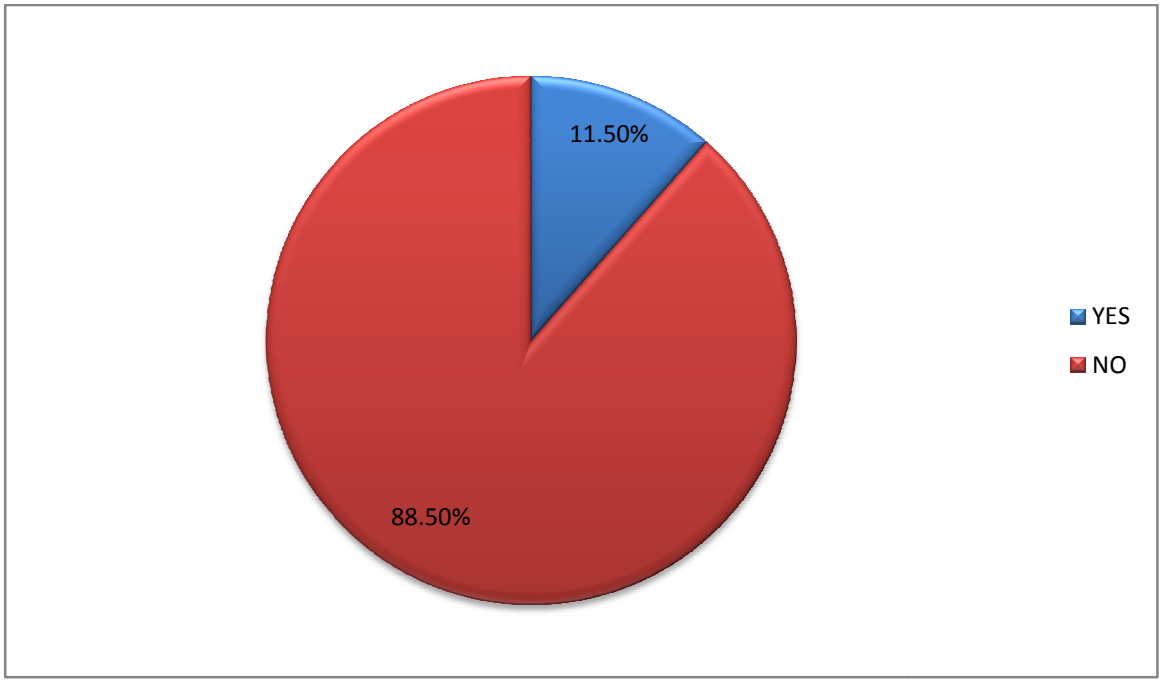
46 cases did not have any birth injuries.

TABLE – 12

BIRTH INJURIES	FREQUENCY	PERCENTAGE
YES	6	11.5%
NO	46	88.5%

FIG – 9

BIRTH INJURIES



FETAL OUTCOME:

Out of the 52 cases included in the study

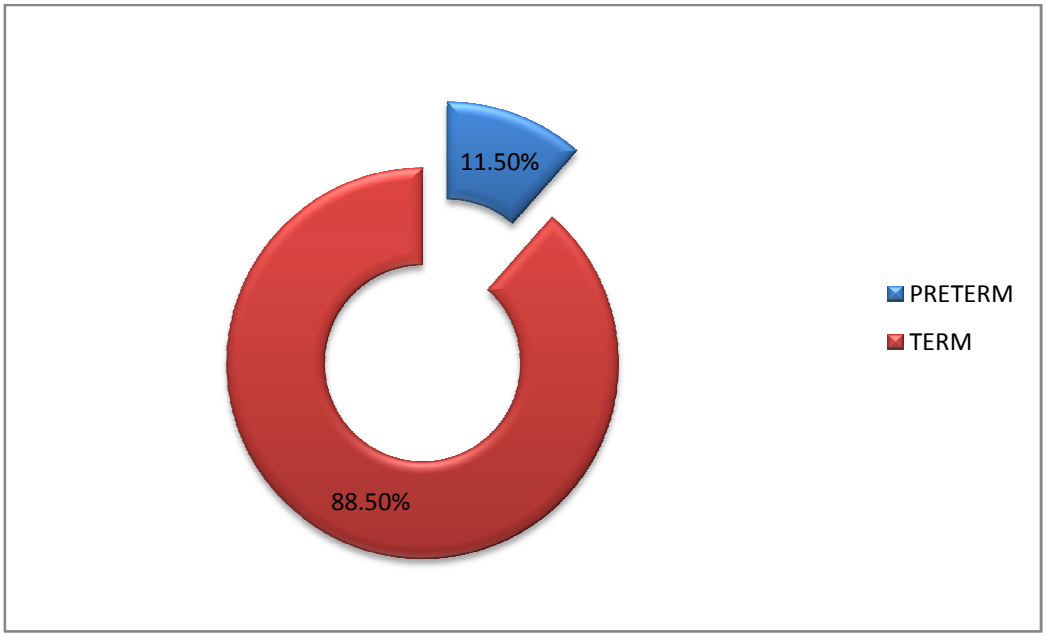
6 babies were born as preterm. Among these 6 babies, 4 babies were born between 34 – 37 weeks and 2 babies were born before 34 weeks.

46 babies were born as term babies.

TABLE – 13

FETAL OUTCOME	FREQUENCY	PERCENTAGE
PRETERM	6	11.5%
TERM	46	88.5%

FIG – 10



GESTATIONAL AGE:

Among the 52 babies studied,

3 babies (5.8 %) were small for gestational age.

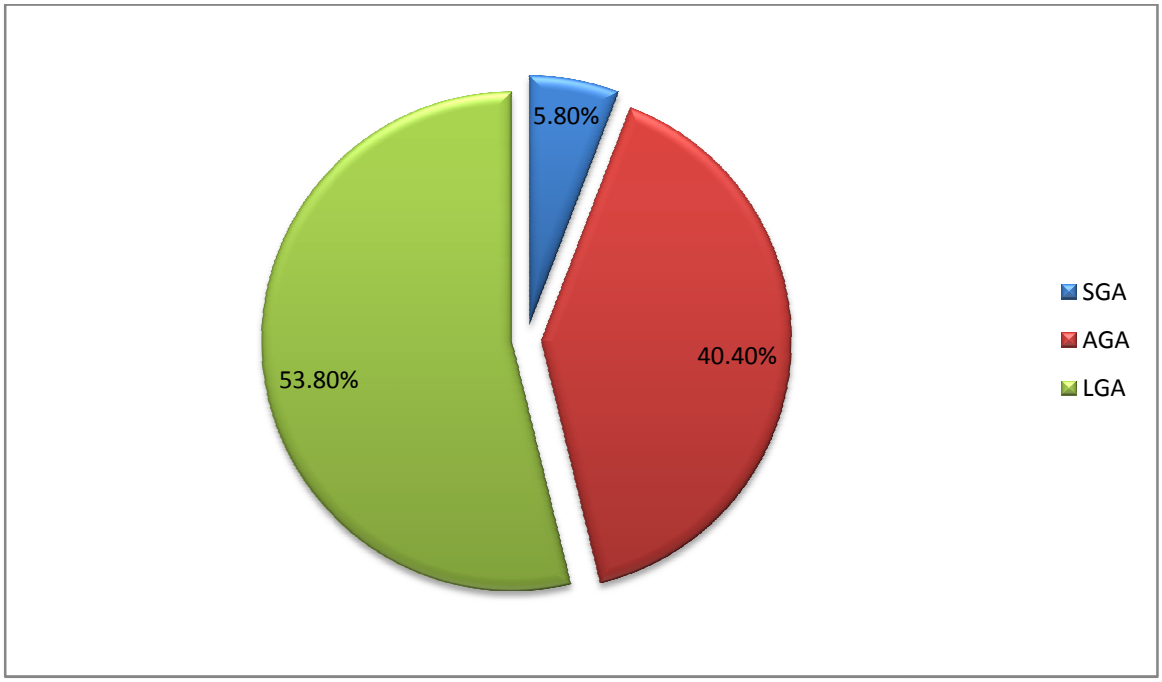
21 babies (40.4 %) were appropriate for gestational age.

28 babies (53.8 %) were large for gestational age.

TABLE – 14

GEST. AGE	FREQUENCY	PERCENTAGE
SGA	3	5.8%
AGA	21	40.4%
LGA	28	53.8%

FIG – 11



MACROSOMIA:

Among the 52 cases included in the study

28 babies (53.8 %) had macrosomia.

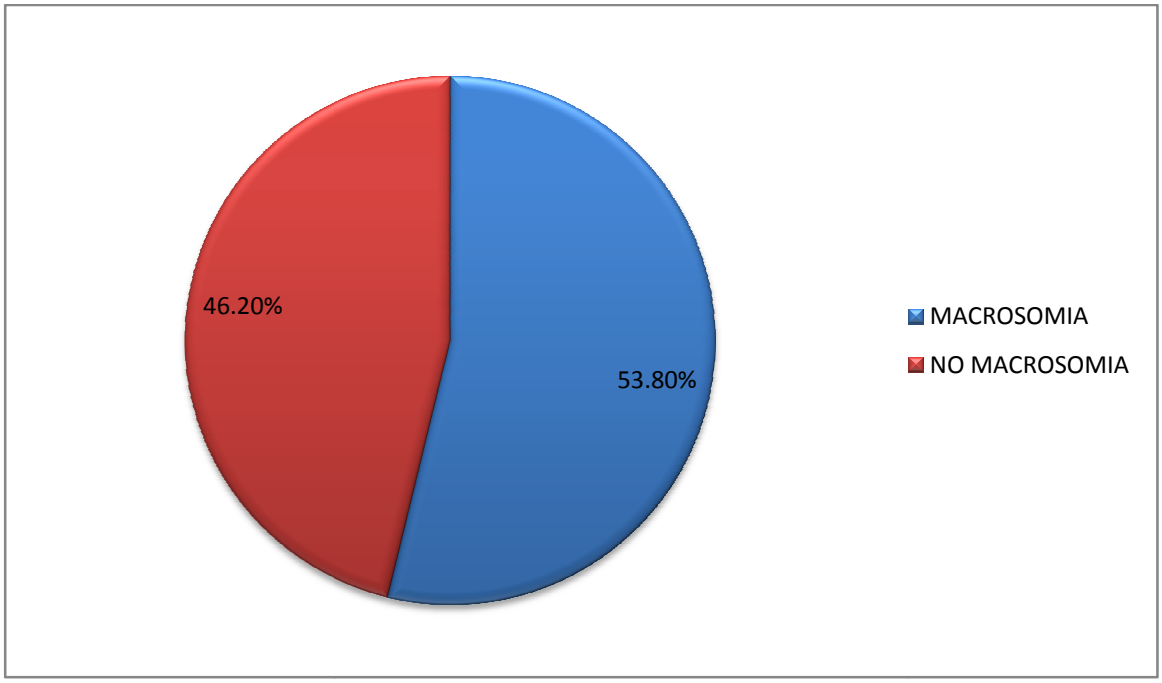
24 babies (46.2 %) did not have macrosomia.

The maximum birth weight among babies was 4.42 kg and the minimum birth weight among babies was 1.62 kg .The mean birth weight was 3.57 kg.

TABLE – 15

MACROSOMIA	FREQUENCY	PERCENTAGE
YES	28	53.8%
NO	24	46.2%

FIG – 12

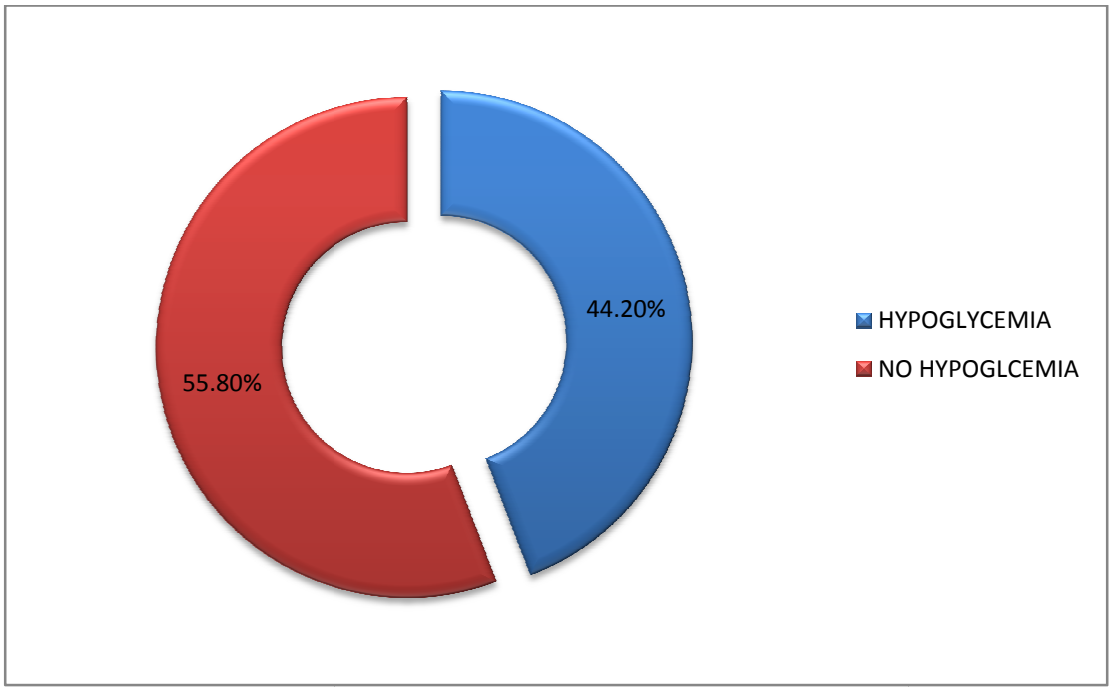


HYPOGLYCEMIA:

In this study, the total no. of cases in which hypoglycemia was documented- 23 cases (44.2%).

The total no. of cases in which hypoglycaemia was not documented- 29 cases (55.8 %).

Fig – 13



In this study, blood sugar values are taken at 1 hr, 2 hr, 3 hr, 6 hr, 9 hr, 12 hr, 24 hr, 36 hr and 48 hrs. The maximum blood sugar value and minimum blood sugar value were noted. Mean blood sugar value was calculated. Most of the hypoglycemic episodes were seen in the first day of life.

VARIOUS BLOOD SUGAR VALUES TAKEN : (TABLE – 16)

BLOOD SUGAR VALUE-AT	MINIMUM VALUE	MAXIMUM VALUE	MEAN VALUE
1 HR	32	97	64.04
2 HR	27	121	67.10
3 HR	22	156	80.10
6 HR	25	158	91.94
12 HR	37	156	98.21
24 HR	71	176	107.19
36 HR	62	168	103.38
48 HR	72	178	111.77

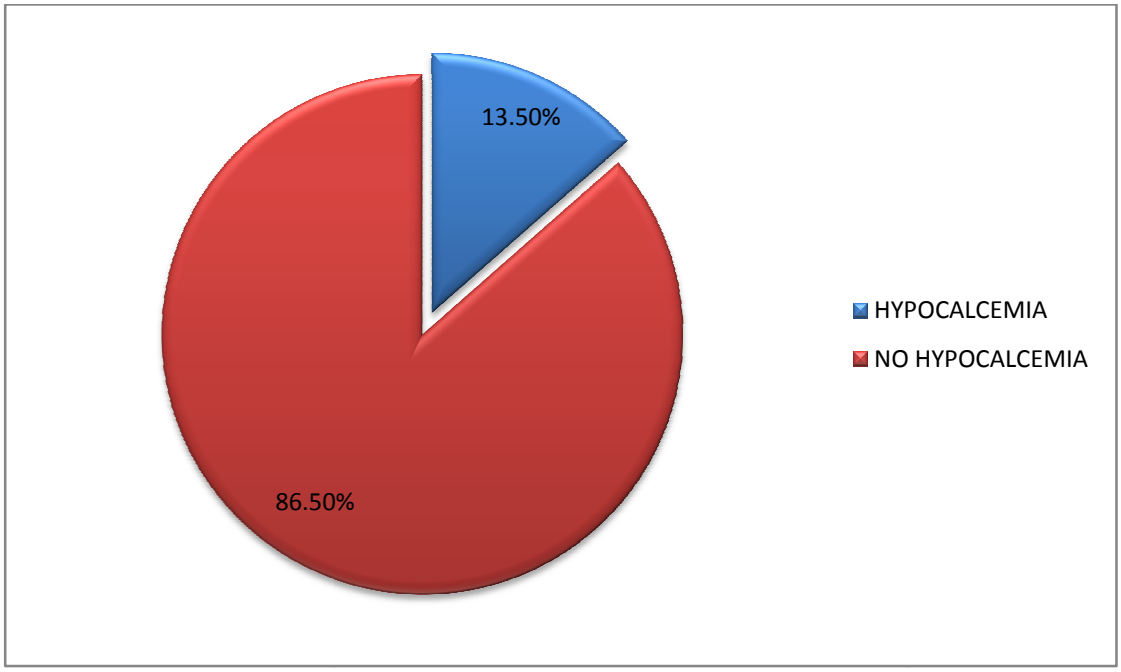
HYPOCALCEMIA:

Among the babies studied, 7 babies (13.5%) had hypocalcemia and 45 babies (86.5 %) did not have hypocalcemia. The maximum value of calcium obtained was 11.56 mg/dl and the minimum value of calcium obtained was 5.24 mg/dl. The mean value of calcium was 9.07.

TABLE – 17

HYPOCALCEMIA	FREQUENCY	PERCENTAGE
YES	7	13.5%
NO	45	86.5%

FIG – 14



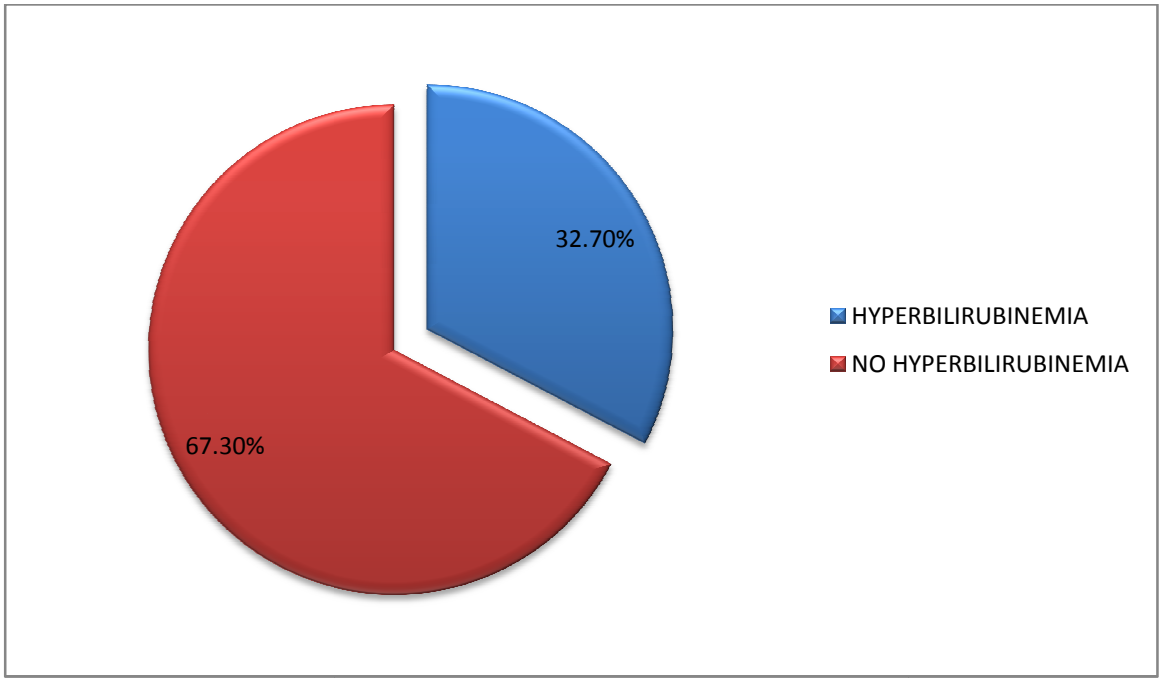
HYPERBILIRUBINEMIA:

In this study, the number of babies who had hyperbilirubinemia was 17 and the number of babies who did not have hyperbilirubinemia was 35.

TABLE – 18

HYPERBILIRUBINEMIA	FREQUENCY	PERCENTAGE
YES	17	32.7%
NO	35	67.3%

FIG – 15



POLYCYTHEMIA:

In this study, among the 52 cases

4 babies (7.7%) had polycythemia.

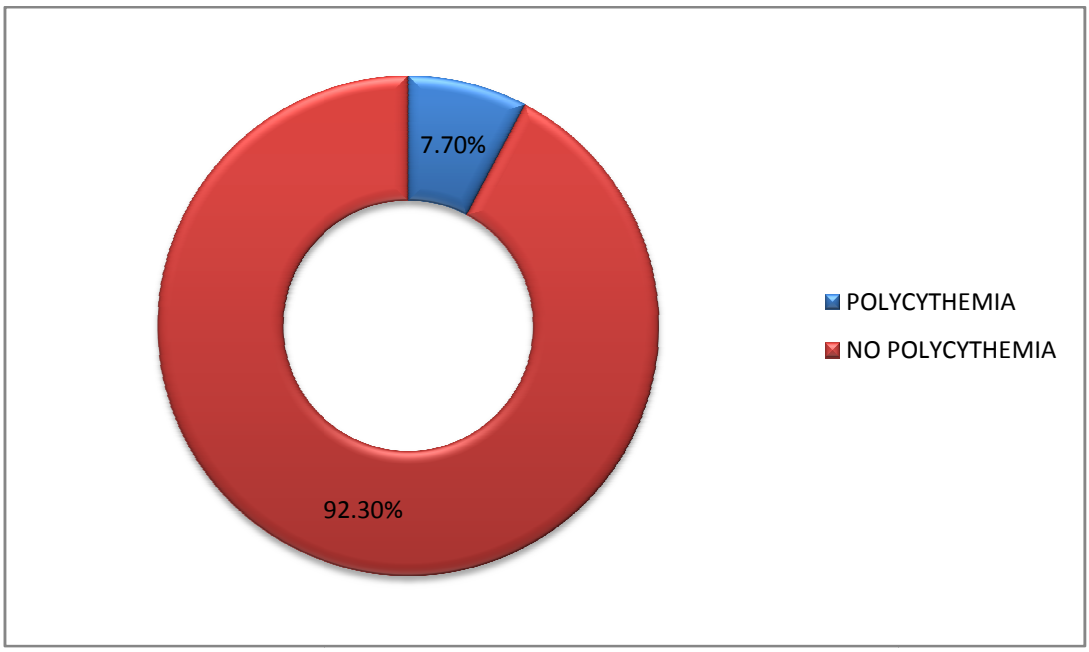
48 babies (92.3%) did not have polycythemia.

The maximum value of PCV was 70.67 and the minimum value of PCV was 46.07.The mean value was 54.43.

TABLE – 19

POLYCYTHEMIA	FREQUENCY	PERCENTAGE
YES	4	7.7%
NO	48	92.3%

FIG – 16



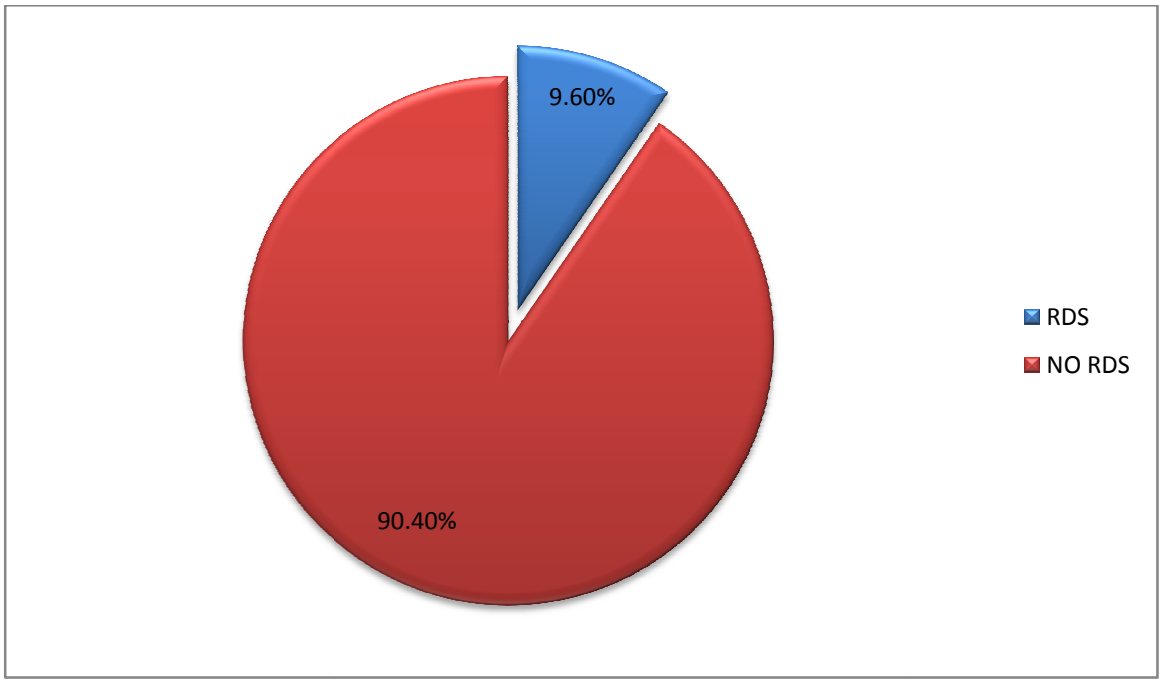
RESPIRATORY DISTRESS SYNDROME:

Among the babies studied, 5 babies (9.6%) had respiratory distress syndrome and 47 babies (90.4%) did not have respiratory distress syndrome. Among the 5 babies 4 were preterm and 1 was a term baby.

TABLE – 20

RDS	FREQUENCY	PERCENTAGE
YES	5	9.6%
NO	47	90.4%

FIG – 17



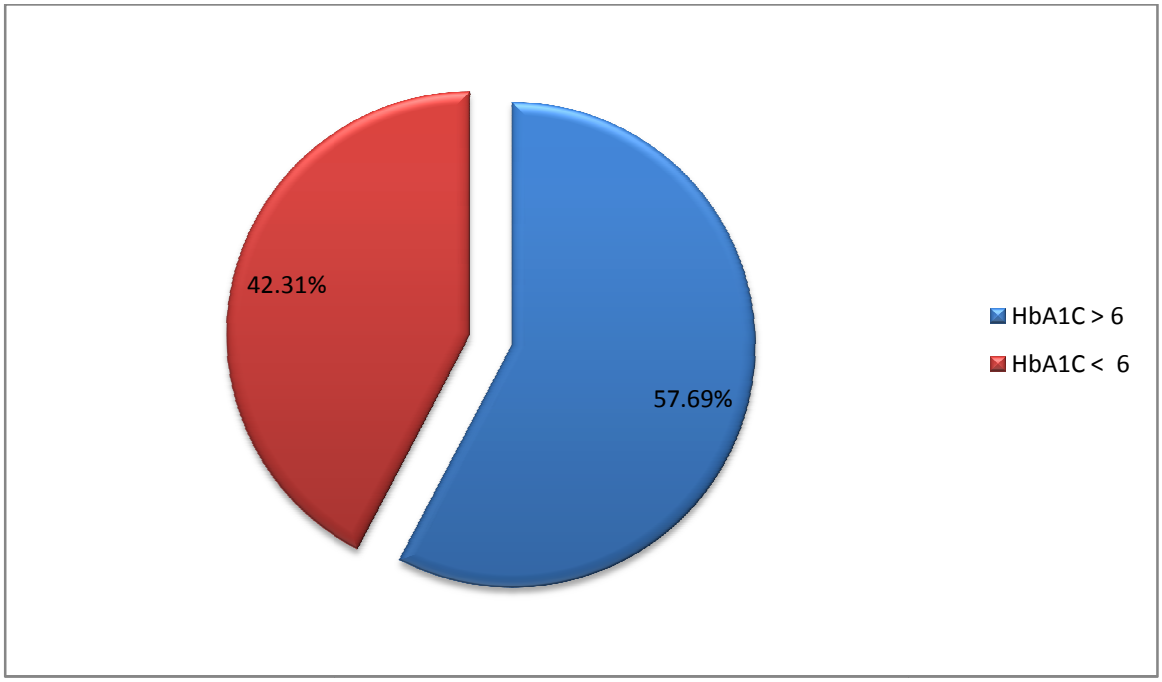
GLYCOSYLATED HAEMOGLOBIN:

Based on the STANDARDS OF MEDICAL CARE IN DIABETES -2012 and GLOBAL GUIDELINES PREGNANCY AND DIABETES, HbA_1C which denotes good glycemic control was taken as < 6. Out of the 52 cases studied, 30 cases had HbA_1C < 6 and 22 cases had HbA_1C > 6.

TABLE – 21

HbA_{1C}	FREQUENCY	PERCENTAGE
< 6	22	42.3%
>6	30	57.7%

FIG – 18



RESULTS

SEX TABLE – 22:

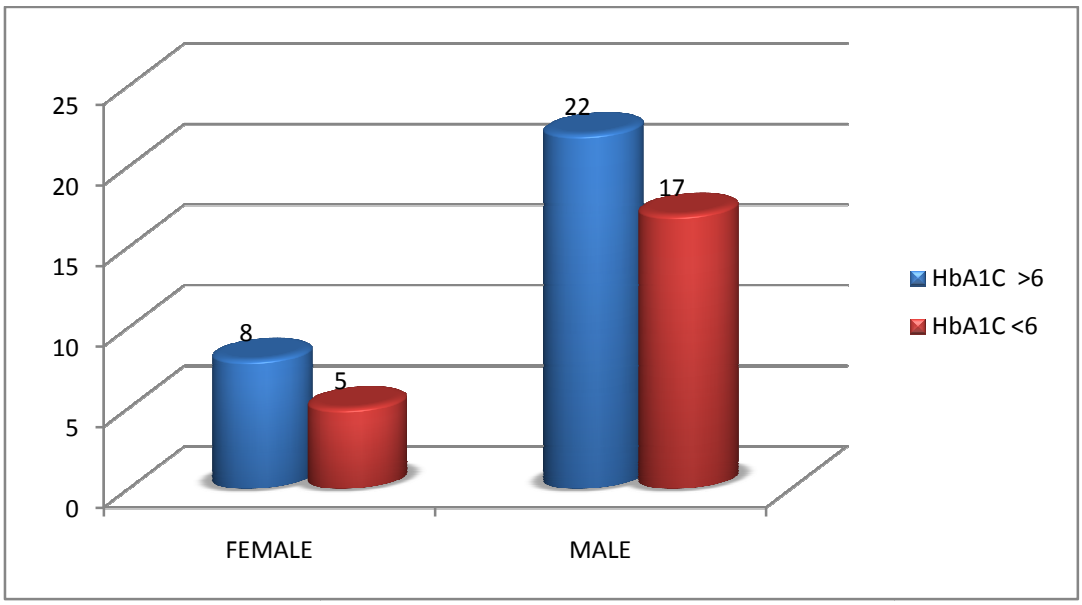
SEX	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
FEMALE	8 (61.5 %)	5 (38.5 %)	13 (100.0 %)
MALE	22 (56.4 %)	17 (43.6 %)	39 (100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

INTERPRETATION :

Among the 30 babies born to mothers with poor glycemic control, 22 babies were males and 8 babies were females.

Among the 22 babies born to mothers with good glycemic control, 17 babies were males and 5 babies were females.

FIG – 19



(P VALUE – 0.746, df – 1)

INFERENCE :

In both the above categories, the number of male babies exceeds the number of female babies. Hence it is convincing that there is no correlation between the sex of the baby born to diabetic mothers and the maternal glycemic control. The statistical analysis done with the above data shows the P value of this variable as 0.746. This was not statistically significant.

BOOKED / UNBOOKED:

TABLE – 23

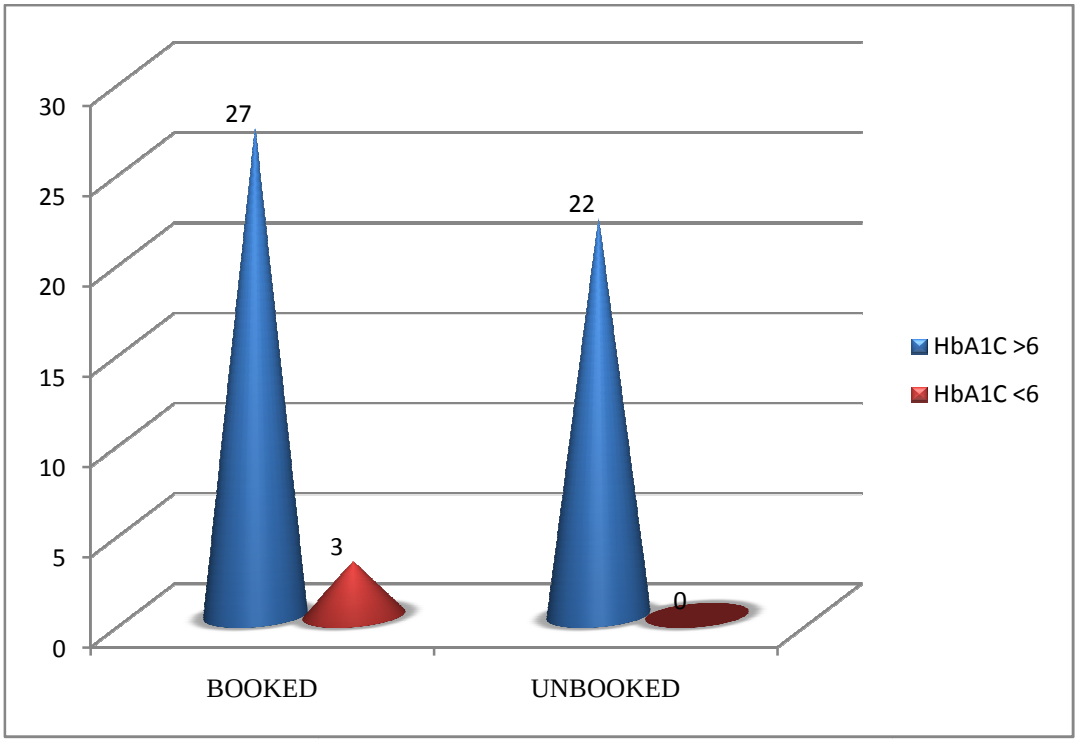
BOOKED	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
YES	27 (55.1 %)	22 (44.9 %)	49 (100.0 %)
NO	3 (100.0 %)	0 (0 %)	3 (100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

INTERPRETATION :

Among the booked cases, 55.1% had higher HbA_1C and 44.9% had lower HbA_1C.

In unbooked cases, 100% of cases had higher HbA_1C values.

FIG – 20



(P VALUE-0.127, df-1)

INFERENCE:

This seems to be highly correlating (Those mothers who had not even booked their pregnancy, would not have gone for regular check up's and would not have taken any treatment to keep their blood sugar under control). But according to the statistical analysis, the P value for this variable is 0.127. This was not statistically significant. This is due to the small sample size.

GDM / DM:

TABLE – 24

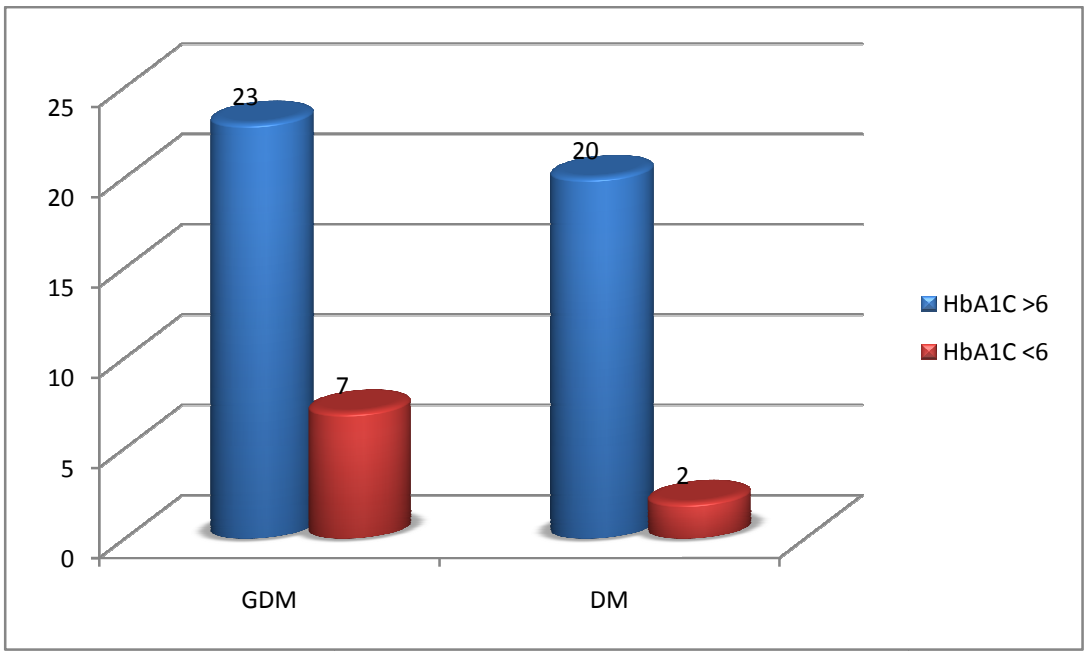
GDM / DM	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
DM	7 (77.8 %)	2 (22.2 %)	9 (100.0 %)
GDM	23 (53.5 %)	20 (46.5 %)	43 (100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

INTERPRETATION :

Among the 9 DM mothers, 7 mothers (77.8 %) had HbA_1C value > 6 and 2 mothers (22.2%) had HbA_1C value < 6.

Among the 43 GDM mothers, 23 mothers (53.5%) had HbA_1C values > 6 and 20 mothers (46.5%) had HbA_1C value < 6.

FIG – 21



(P VALUE – 0.180, df-1)

INFERENCE :

Here it seems that whether DM or GDM, the number of cases with HbA_1C value which implies poor glycemic control exceeds the number of cases with HbA_1C value which implies good glycemic control. This indicates that the antenatal care is not adequate and also it indicates that the mothers are not aware of the complications associated with poor glycemic control during pregnancy. But when analysed, the P value for this variable was 0.180.

This was not statistically significant.

COMPLIANCE:

TABLE – 25

COMPLIANCE	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
COMPLIANT	**16 (43.2 %)**	**21 (56.8 %)**	**37 (100.0 %)**
NOT COMPLIANT	**9 (90.0 %)**	**1 (10.0 %)**	**10 (100.0 %)**
NOT APPLICABLE	**5 (100.0 %)**	**0 (0 %)**	**5 (100.0 %)**
TOTAL	**30 (57.7 %)**	**22 (42.3 %)**	**52 (100.0 %)**

INTERPRETATION :

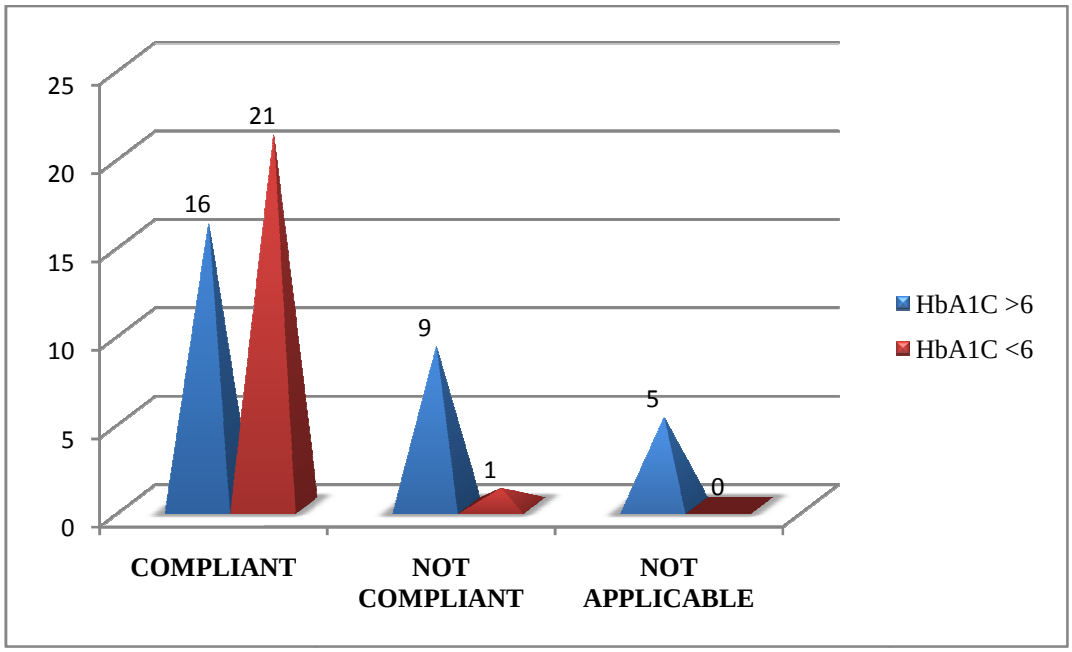
Here, among those patients who were compliant with treatment, 43.2% had higher HbA_1C values and 56.8% had lower HbA_1C values.

Among those patients who were not compliant 90% had higher HbA_1C values and 10% had lower HbA_1C values.

Not applicable implies those patients who were not treated. Among them 100% had higher HbA_1C values.

FIG – 22

COMPLIANCE



(P VALUE-O.OO4, df-2)

INFERENCE :

Here it shows that those mothers who were compliant had HbA_1C values < 6, which indicates good glycemic control. The P value of this variable was 0.004. Statistically this observation was found to be highly significant.

TREATMENT:

TABLE – 26

TREATMENT	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
DIET	9 (47.4 %)	10 (52.6 %)	19 (100.0 %)
INSULIN	16 (57.1 %)	12 (42.9 %)	28 (100.0 %)
NOT TREATED	5 (100.0 %)	0 (0 %)	5 (100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

INTERPRETATION :

According to the treatment given, the patients were divided into 3 groups.

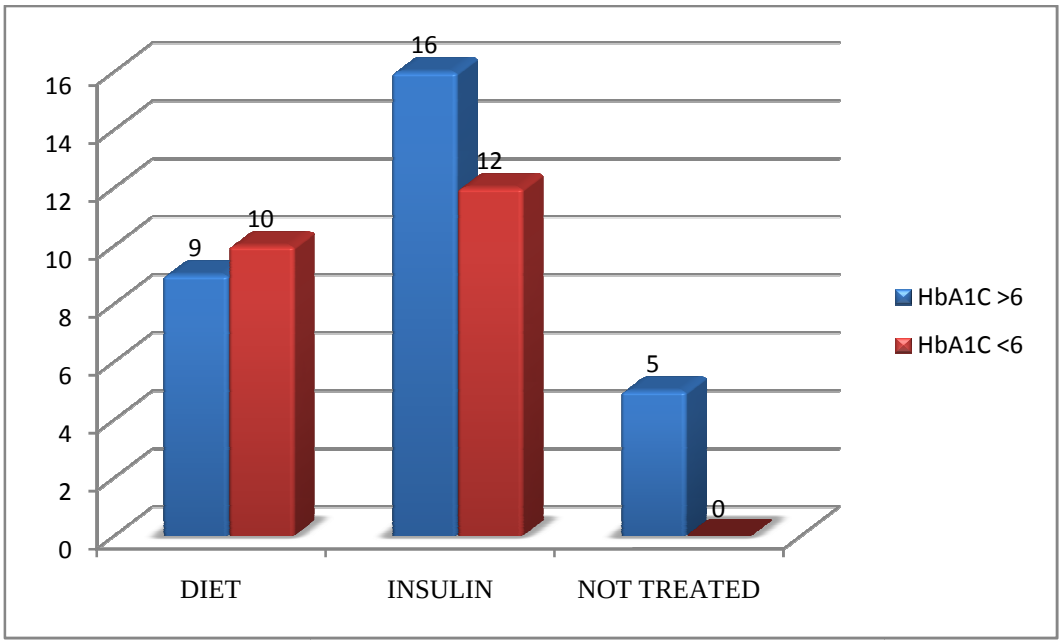
In patients who were treated with diabetic diet, 47.4% cases had HbA_1C values >6 and 52.6% cases had HbA_1C values <6.

In patients who were treated with insulin, 57.1% cases had HbA_1C values >6 and 42.9% cases had HbA_1C values <6.

In patients who were not treated, 100% of cases had HbA_1C values >6.

FIG – 23

TREATMENT



(P VALUE-0.105, df-2)

INFERENCE :

Among the mothers who were on diabetic diet, about half of them had poor glycemic control. Among those mothers who were on insulin, more no of mothers had poor glycemic control. This may be due to the fact that outcome depends on various other factors like compliance.

Among the non treated mothers, all of them had poor glycemic control. This seems to be correlating. However, the results were not statistically significant. The P value was 0.105.

MODE OF DELIVERY:

TABLE – 27

DELIVERY	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
FORCEPS	1 (50.0 %)	1 (50.0 %)	2 (100.0 %)
LN	3 (50.0 %)	3 (50.0 %)	6 (100.0 %)
LSCS	26 (59.1 %)	18 (40.9 %)	44 (100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

INTERPRETATION:

In patients with HbA_1C value >6,

3.3% cases were delivered by forceps.

10% cases were delivered by labour naturalis.

86.6% cases were delivered by caesarean section.

In patients with HbA_1C value< 6,

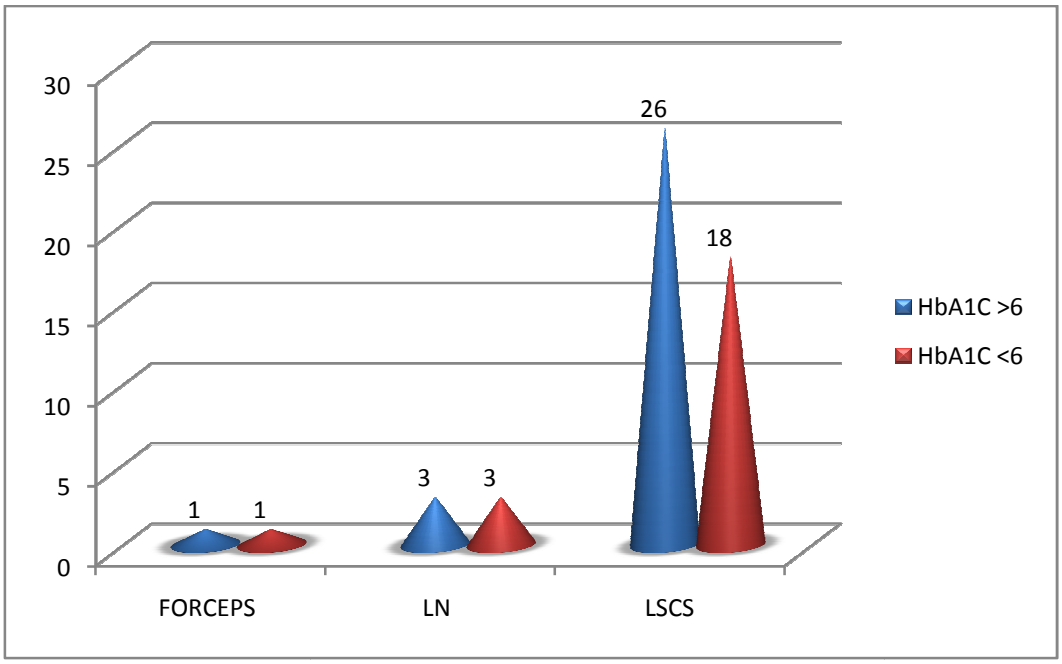
4.5% cases were delivered by forceps.

13.6% cases were delivered by labour naturalis.

81.8% cases were delivered by caesarean section.

FIG – 24

MODE OF DELIVERY



(P VALUE-O.892, df-2)

INFERENCE :

This shows that most of the diabetic pregnancies are delivered by caesarean section .This seems to be a good approach as the incidence of birth injuries in macrosomic babies could be avoided.

This was not statistically significant.

The P value was 0.892.

ASPHYXIA:

TABLE – 28

ASPHYXIA	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
YES	6 (66.7 %)	3 (33.3 %)	9 (100.0 %)
NO	24 (55.8 %)	19 (44.2 %)	43(100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

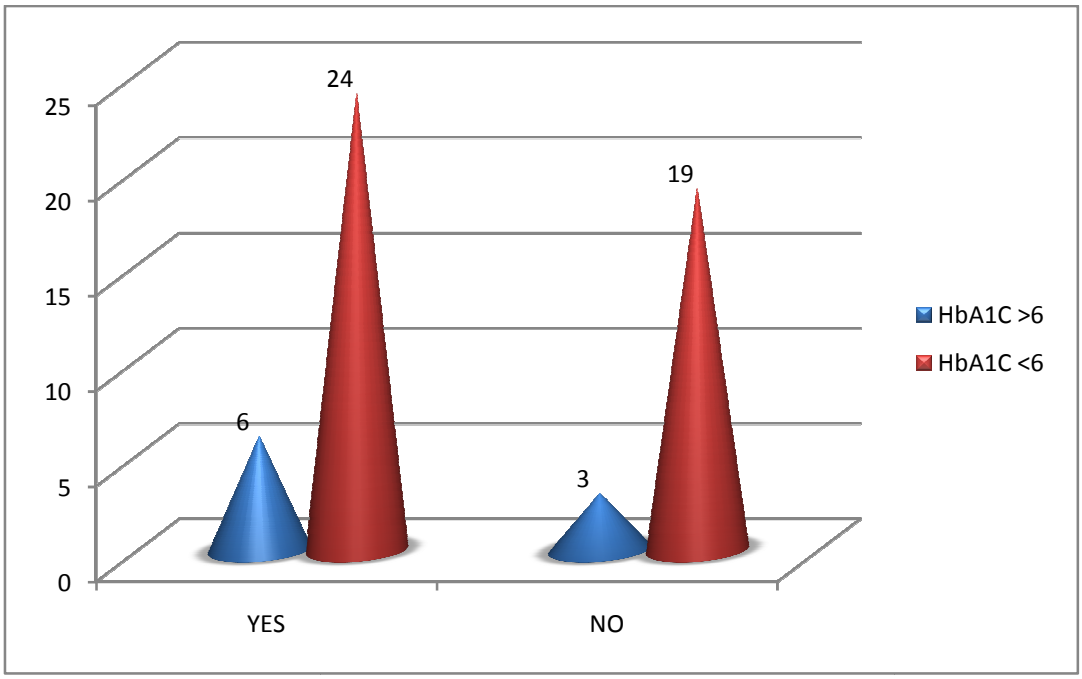
INTERPRETATION :

Among the 9 babies who had experienced birth asphyxia, 6 babies (66.7%) were born to mothers with poor glycemic control and 3 babies (33.3%) were born to mothers with good glycemic control.

Among the 43 babies who did not experience birth asphyxia, 24 babies (55.8%) were born to mothers with poor glycemic control and 19 babies (44.2%) were born to mothers with good glycemic control.

FIG – 25

ASPHYXIA



(P VALUE – 0.549, df – 1)

INFERENCE :

Here among the babies who had experienced birth asphyxia, most of them were born to diabetic mothers with poor glycemic control. But among the babies who did not have birth asphyxia also 24 babies were born to mothers with poor glycemic control. This might be due to the more no of caesarean deliveries conducted in diabetic pregnancies. This was not statistically significant. The P value was 0.549.

BIRTH INJURIES:

TABLE – 29

INJURIES	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
YES	4 (66.7 %)	2 (33.3 %)	6 (100.0 %)
NO	26 (56.5 %)	20 (43.5 %)	46 (100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

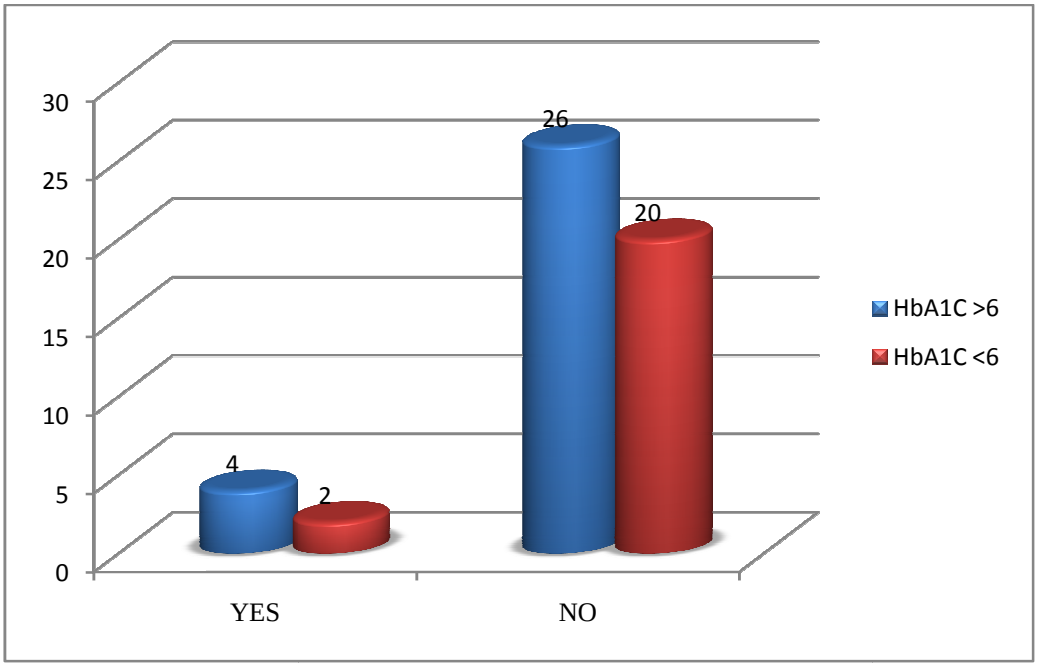
INTERPRETATION :

Out of the 6 babies who had birth injuries in this study, 4 babies (66.7%) were born to mothers with poor glycemic control and 2 babies (33.3%) were born to mothers with good glycemic control.

Out of the 46 babies who did not have any birth injuries, 26 babies (56.5%) were born to mothers with poor glycemic control and 20 babies (43.3%) were born to mothers with good glycemic control.

FIG – 26

BIRTH INJURIES



(P VALUE – 0.636, df – 1)

INFERENCE :

In this study among the babies who had birth injuries, majority of them were born to diabetic mothers with poor glycemic control. This implies that a good glycemic control is needed to avoid birth injuries in babies born to diabetic mothers. But among the other group without birth injuries, 26 babies were born to mothers with poor glycemic control. This might be due to the more number of caesarean deliveries conducted in diabetic pregnancies. This was not statistically significant.

The P value was 0.549.

GESTATIONAL AGE:

TABLE – 30

GESTATIONAL AGE	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
SGA	0 (0 %)	3 (100.0 %)	3 (100.0 %)
AGA	4 (19.0)	17 (81.0 %)	21 (100 .0 %)
LGA	26 (92.9 %)	2 (7.1 %)	28 (100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

INTERPRETATION :

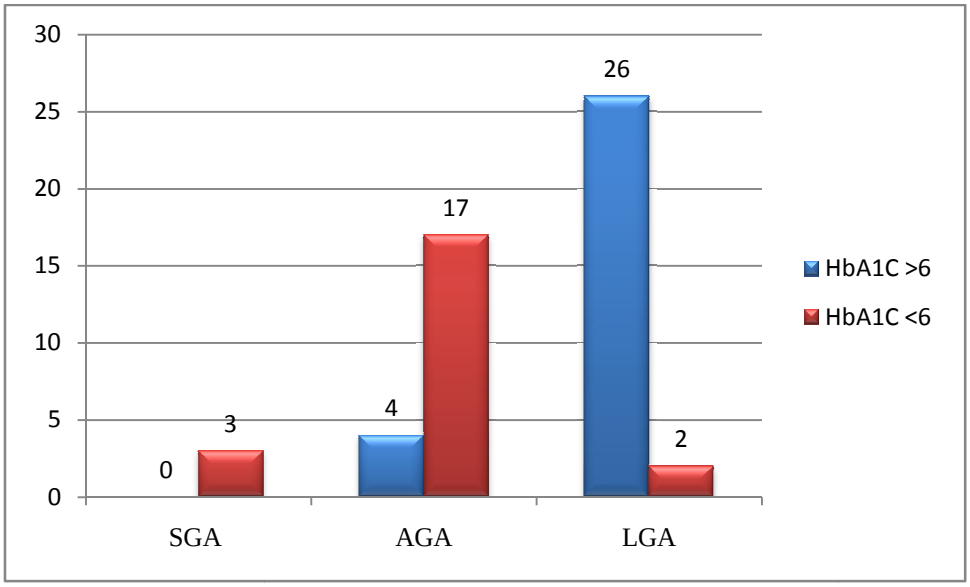
Among the 3 SGA babies in this study, all the babies were born to mothers with good glycemic control.

Among the 21 AGA babies in this study, 4 babies (19.0%) were born to mothers with poor glycemic control and 17 babies (81.0%) were born to mothers with good glycemic control.

Among the 28 LGA babies in this study, 26 babies (92.9%) were born to mothers with poor glycemic control and 2 babies (7.1%) were born to mothers with good glycemic control.

FIG – 27

GESTATIONAL AGE



(P VALUE – 0.000, df- 2)

INFERENCE :

From the above data it is clear that most of the LGA babies in this study were born to diabetic mothers with poor glycemic control. It implies that a good glycemic control is essential to have AGA baby. This was found to be statistically highly significant.

The P value was 0.000.

FETAL OUTCOME:

TABLE – 31

FETAL OUTCOME	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
PRETERM	2 (33.3%)	4 (66.7%)	6 (100.0%)
TERM	29 (63.0%)	17 (37.0%)	46 (100.0%)
TOTAL	30 (57.7%)	22 (42.3%)	52 (100.0%)

(P VALUE – 0.031, df – 1)

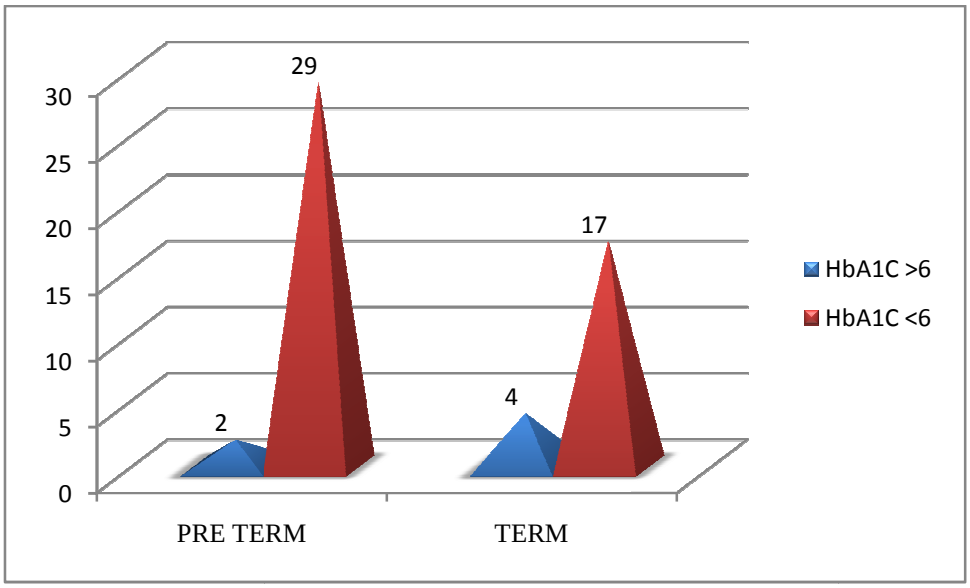
INTERPRETATION :

Among the 6 preterm babies in this study, 4 babies (66.7 %) were born to mothers with good glycemic control and 2 babies (33.3 %) were born to mothers with poor glycemic control.

Among the 46 term babies in this study, 29 babies (63.0%) were born to mothers with poor glycemic control and 17 babies (37.0 %) were born to mothers with good glycemic control.

FIG – 28

TERM/ PRE TERM



INFERENCE :

From the above analysis it is clear that there is no correlation between preterm deliveries and maternal glycemic control. There are a lot of causes for preterm deliveries of which Diabetes in pregnancy is one. Hence the incidence of preterm deliveries could not be prevented by strict maternal glycemic control alone. However the results were not statistically significant.

The P value was 0.064.

MACROSOMIA

TABLE – 30

MACROSOMIA	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
YES	26 (92.9 %)	2 (7.1 %)	28 (100.0 %)
NO	4 (16.7 %)	20 (83.3 %)	24 (100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

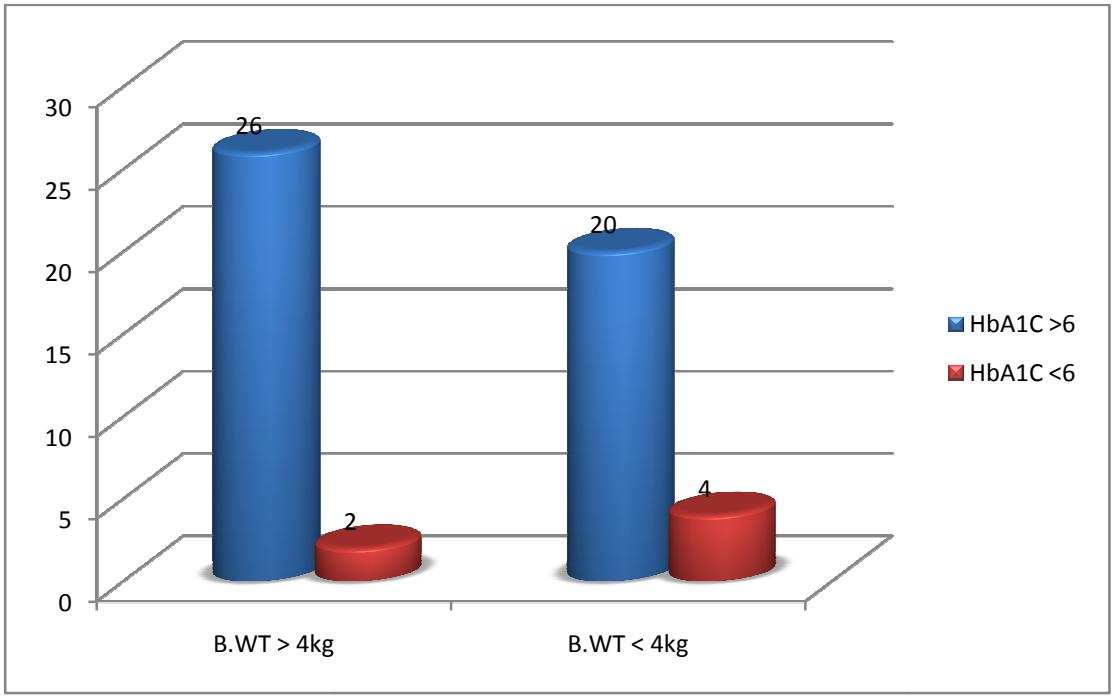
INTERPRETATION :

Out of the 28 macrosomic babies in this study, 26 babies (92.9%) were born to mothers with poor glycemic control and 2 babies (7.1%) were born to mothers with good glycemic control.

Out of the 24 babies without macrosomia in this study, 20 babies (83.3%) were born to mothers with good glycemic control and 4 babies (16.7%) were born to mothers with poor glycemic control.

FIG – 27

BIRTH WEIGHT



(P VALUE – 0.000, df – 1)

INFERENCE :

Here most of the macrosomic babies were born to diabetic mothers with poor glycemic control. Macrosomic babies are usually associated with other complications such as birth asphyxia and birth injuries. Hence it indicates that it is mandatory to have a good glycemic control during pregnancy to avoid the complication of macrosomia. The results were found to be statistically highly significant.

The P value was 0.000.

HYPOGLYCEMIA:

TABLE – 31

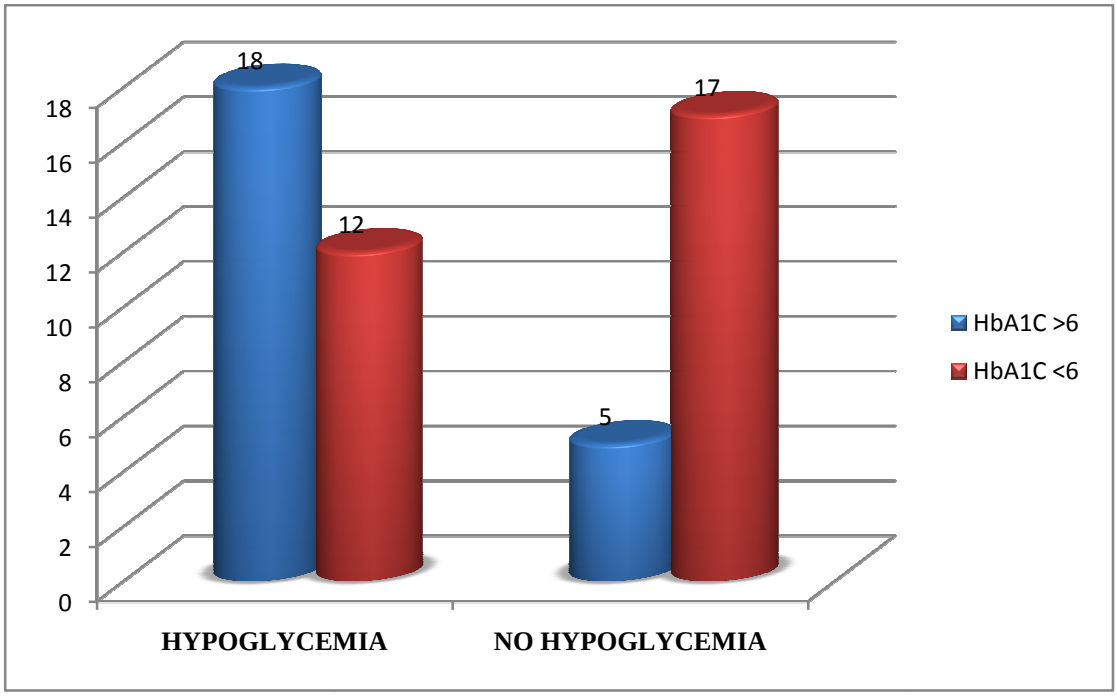
HYPOGLYCEMIA	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
YES	18 (78.3 %)	5 (21.7 %)	23 (100.0 %)
NO	12 (41.4 %)	17 (58.6 %)	29 (100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

INTERPRETATION :

Out of the 23 babies with hypoglycemia in this study, 18 babies (78.3%) were born to mothers with poor glycemic control and 5 babies (21.7%) were born to mothers with good glycemic control.

Out of the 29 babies without hypoglycemia in this study, 17 babies (58.6%) were born to mothers with good glycemic control and 12 babies (41.4%) were born to mothers with poor glycemic control.

FIG – 28



(P VALUE – 0.008, df – 1)

INFERENCE :

With the above data it is clear that hypoglycemia in neonates born to diabetic mothers is more common in pregnancies without good glycemic control. Prolonged hypoglycemia is associated with poor neuro developmental outcome. Hypoglycemia may even led to death of the infant if not corrected promptly. This emphasizes the need for good glycemic control during pregnancy to avoid this complication in newborn born to diabetic mothers. This was found to have statistical significance. The P value was 0.008.

HYPOCALCEMIA:

TABLE – 32

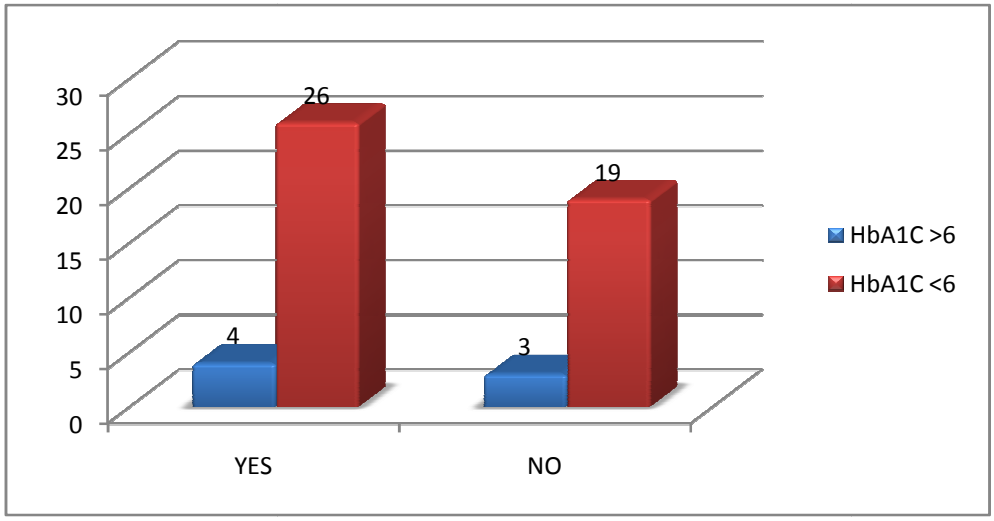
HYPOCALCEMIA	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
YES	4 (57.1 %)	3 (42.9 %)	7 (100.0 %)
NO	26 (57.8 %)	19(42.2 %)	45 (100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

INTERPRETATION :

Among the 7 babies who had hypocalcemia in this study, 4 babies (57.1%) were born to mothers with poor glycemic control and 3 babies (42.9%) were born to mothers with good glycemic control.

Among the 45 babies without hypocalcemia in this study, 26 babies (57.8%) were born to mothers with poor glycemic control and 19 babies (42.2%) were born to mothers with good glycemic control.

FIG – 29
HYPOCALCEMIA



(P VALUE - 0.975, df – 1)

INFERENCE :

The above interpretation shows that there is no definite correlation between incidences of hypocalcemia in neonates born to diabetic mothers with maternal glycemic control. This is because hypocalcemia in newborn is influenced by various other factors too. This was not statistically significant.

The P value was 0.975.

HYPERBILIRUBINEMIA:

TABLE – 33

HYPERBILIRUBINEMIA	**HbA_1C CATEGORY**		**TOTAL**
	HIGHER (> 6)	**LOWER (< 6)**	
YES	7 (41.2 %)	10 (58.8 %)	17 (100.0 %)
NO	23 (65.7 %)	12 (34.3 %)	35 (100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

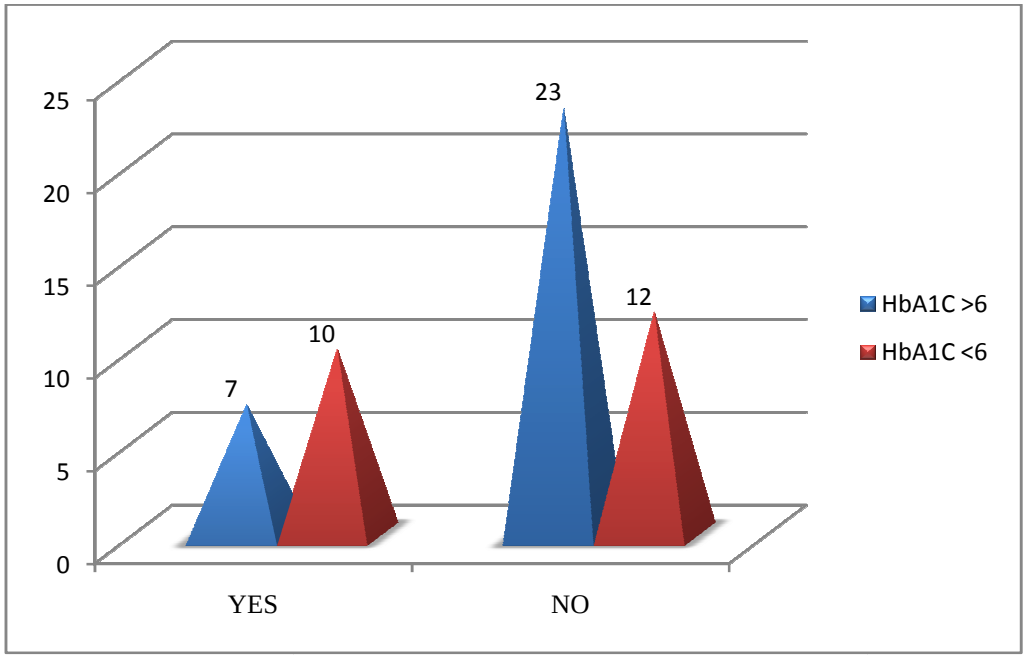
INTERPRETATION :

In this study, among the 17 babies who had hyperbilirubinemia, 10 babies (58.8%) were born to mothers with good glycemic control and 7 babies (41.2%) were born to mothers with poor glycemic control.

Among the 35 babies who did not have hyperbilirubinemia, 23 babies (65.7%) were born to mothers with poor glycemic control and 12 babies (34.3%) were born to mothers with good glycemic control.

FIG – 30

HYPERBILIRUBINEMIA



(P VALUE – 0.093, df – 1)

INFERENCE :

From the above details it is clear that there is no correlation for the above variables. This is because the reasons for hyperbilirubinemia in newborn are various. There were many other causes for hyperbilirubinemia in this study, (e.g.) 2 babies had ABO incompatibility and one baby had RH incompatibility.

This was not statistically significant.

The P value was 0.093.

POLYCYTHEMIA:

TABLE – 34

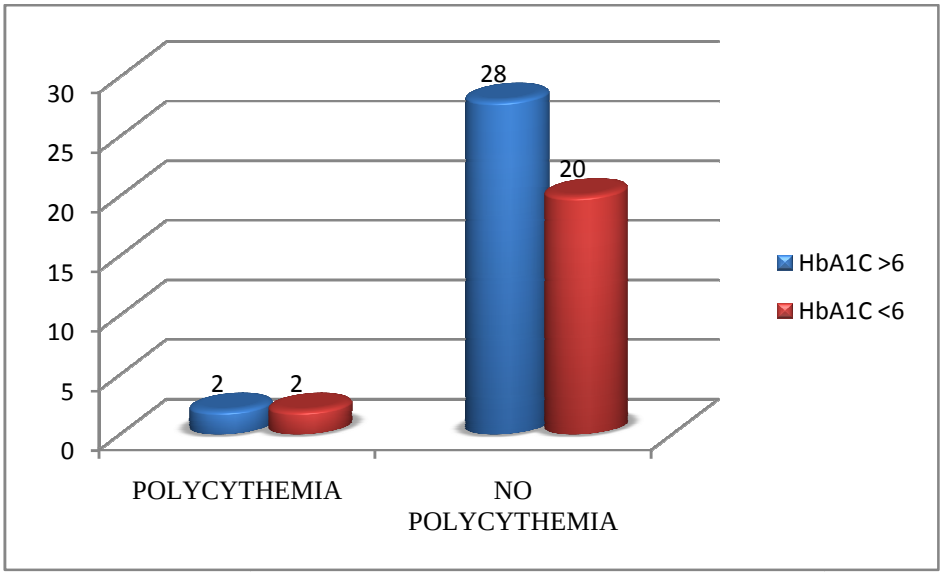
POLYCYTHEMIA	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
YES	2 (50.0 %)	2 (50.0 %)	4 (100.0 %)
NO	28 (58.3 %)	20 (41.7 %)	48 (100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

INTERPRETATION :

Out of the 4 babies who had polycythemia in this study, 2 babies (50.0%) were born to mothers with poor glycemic control and 2 babies (50.0%) were born to mothers with good glycemic control.

Out of the 48 babies who did not have polycythemia in this study, 28 babies (58.3%) were born to mothers with poor glycemic control and 20 babies (41.7%) were born to mothers with good glycemic control.

FIG – 31



(P VALUE – 0.746, df – 1)

INFERENCE :

The data given above shows that there is no correlation between polycythemia in newborn and maternal blood sugar. This is because polycythemia in newborn is influenced by many factors. In this study among the 4 cases with polycythemia, 1 was an IUGR baby. This was not statistically significant. The P value was 0.746.

RESPIRATORY DISTRESS SYNDROME:

TABLE – 35

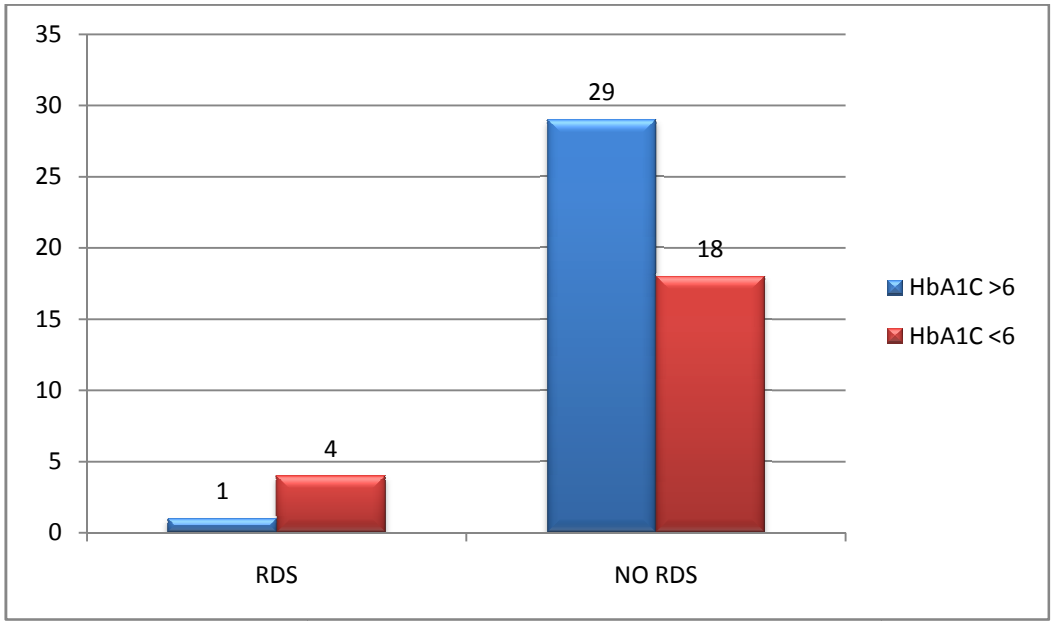
RDS	HbA_1C CATEGORY		TOTAL
	HIGHER (> 6)	LOWER (< 6)	
YES	1 (20.0 %)	4 (80.0 %)	5 (100.0 %)
NO	29 (61.7 %)	18 (38.3 %)	47 (100.0 %)
TOTAL	30 (57.7 %)	22 (42.3 %)	52 (100.0 %)

INTERPRETATION :

Among the 5 babies who had respiratory distress syndrome in this study, 4 babies (80.0%) were born to mothers with good glycemic control and only one baby (20.0%) was born to mother with poor glycemic control.

Among the 47 babies who did not have respiratory distress syndrome in this study, 29 babies (61.7%) were born to mothers with poor glycemic control and 18 babies (38.3%) were born to mothers with good glycemic control.

FIG – 32



(P VALUE – 0.073, df – 1)

INFERENCE :

The above analysis shows that there is no correlation between the incidence of respiratory distress syndrome in neonates born to diabetic mothers and maternal glycemic control. This is due to the various causes of respiratory distress syndrome in newborn. All the 5 babies with respiratory distress syndrome in this study were delivered as preterm. This was not statistically significant. The P value was 0.073.

DISCUSSION

Diabetes mellitus complicating pregnancy is a global problem and this disease progression is in an increasing trend. Among the pregnancies that are complicated by diabetes, 80% accounts for gestational diabetes and 20% accounts for pre gestational diabetes. The complications that occur, both in diabetic mothers during pregnancy and neonates born to them are unique[39]. Various studies are being conducted worldwide on the complications of diabetic pregnancies, and more changes are being made in the treatment modalities for a fine tuning. Both the treating obstetricians and paediatricians should be familiar with the newer guidelines in the management of diabetes complicating pregnancies.

Here an attempt was made to describe the pattern of complications in newborn born to diabetic mothers and emphasize the need for a strict glycemic control during pregnancy and careful follow up of newborn during the early neonatal period for early diagnosis of the complications.

Totally 52 babies born to diabetic mothers and admitted in Tirunelveli medical college hospital nursery during the study period was analysed. According to the American diabetic association guidelines, diabetic mothers were categorised as 2 groups

Mothers with HbA_1C values < 6.

Mothers with HbA_1C values > 6.

The complications in newborn delivered by these two groups of mothers were compared and analysed to indicate the impact of maternal glycemic levels on their newborn. In this study out of the 52 cases, 30 cases had $HbA_1C < 6$ and 22 cases had $HbA_1C > 6$. The maximum HbA_1C value was 11.46 and the minimum HbA_1C value was 4.51. The mean HbA_1C value was 7.52.

AGE

Most of the babies in the present study were included on day 1 of life as the blood sugar values were taken starting from first hour of life. Only one baby was included on the second day as it was a referred case. In other studies referred, age of the mothers had been taken as a variable.

SEX

In this study among the babies born, 75% were boys and 25% were girls. There is no difference in the incidence of complications between boys and girls. Also, there is no predilection for either sex in both group of mothers included in the study. The sex of the baby born is not related to the glycemic values of the mother.

GDM / DM

TABLE – 38

STUDIES	GDM	DM
Aarumugam et al[40]	92.7 %	7.3 %
Banerjee et al[41]	73.3%	26.6%
Kavitha et al[42]	64.8 %	35.1 %
Akhlagi et al	27.0 %	73.0 %
Abdulbari et al	16.3 %	83.7 %
My study	82.7 %	17.3 %

In most of the studies the percentage of GDM mothers is greater than the percentage of DM mothers. In this study too, it is the same. But in the studies from Middle East countries, the incidence of DM is more than GDM. This might be due to their dietary habits and their lifestyle.

TREATMENT

In the present study, 36.5 % of mothers were treated with diabetic diet, 53.8 % of mothers were treated with insulin and 9.6 % were not treated. In study done by Alam et al[43] at National Institute of Child Health, Karachi (Aug 1999 to Jan 2000), 12.5 % of mothers were treated with diabetic diet, 47.5 % of mothers were treated with insulin and 40.0 % were not treated. This implies that more number of mothers are attending antenatal check-up regularly and are compliant in our place.

MODE OF DELIVERY

In this study 84.6 % of babies were delivered by LSCS, 11.5 % of babies were delivered by LN and 3.8 % of babies were delivered by forceps. In Alam et al study 55.0 % of babies were delivered by LSCS and 45.0 % of babies were delivered by LN. In Akhlagi et al[44] study conducted at Mashad university of medical sciences, Iran (Jan 2001 – April 2002), 79.8 % of babies were delivered by LSCS and 20.2 % of babies were delivered by LN. This is comparable with the present study.

ASPHYXIA

In this study 17.3 % of babies experienced asphyxia. In Alam et al study conducted at National Institute of Child Health, Karachi 15.0 % in the study has experienced asphyxia. This is comparable with the present study.

BIRTH INJURIES

In this study 11.5 % of babies had birth injuries. In a study by Abdul et al[45] conducted at antenatal clinics of women's hospital, Qatar, 11.0 % of babies had birth injuries. In Alam et al study 17.5 % of babies had birth injuries. This is comparable with the present study.

FETAL OUTCOME

TABLE – 39

STUDIES	PRETERM	TERM
Getu et al	25.0 %	75.0 %
Akhlagi et al	29.4 %	70.6 %
Abdul et al	20.9 %	79.1 %

In this study 11.5 % of babies were born as preterm and 88.5 % of babies were born as term. This is comparable with the above studies.

GESTATIONAL AGE

In this study 53.8 % of babies were LGA, 40.4 % of babies were AGA and 5.8 % of babies were SGA. In Alam et al study 45.0 % of babies were LGA, 50.0 % of babies were AGA and 5.0 % of babies were SGA. This is comparable with the present study.

HYPOGLYCEMIA

In this study 44.2 % of babies had hypoglycemia. In Akhlagi et al study 50.4 % of babies had hypoglycemia. In Alam et al study 35.0 % of babies had hypoglycemia. This is comparable with the present study.

HYPOCALCEMIA

In this study 13.5 % of babies had hypocalcemia. In Alam et al study 15.0 % of babies had hypocalcemia. This is comparable with the present study.

HYPERBILIRUBINEMIA

In this study 32.7 % of babies had hyperbilirubinemia. In Alam et al study 30.0 % of babies had hyperbilirubinemia. This is comparable with the present study.

LIMITATIONS OF THE STUDY

1. Complications due to impaired first trimester glycemic control such as congenital malformations were not included in this study.
2. Incidence of IUD, Abortion and still born were not taken in this study.
3. Incidence of Hypertrophic cardiomyopathy, which occurs due to poor glycemic control in the third trimester, could not be found out because of the non availability of echocardiography.
4. Another third trimester complication, Lazy left colon syndrome was not included in this study.
5. Foetal insulin levels in the cord blood sample to confirm hyperinsulinemia as the cause for the complications was not done.
6. The sample size of this study was small and it was difficult to compare the complications in mothers with good glycemic control against the complications in mothers with poor glycemic control.

CONCLUSION

Complications in newborn born to diabetic mothers are an emerging but preventable problem that seeks more importance.

Gestational diabetes is more common than pregestational diabetes.

The babies included in the present study had significant number of complications especially in neonates born to mothers with poor glycemic control.

Diabetic mothers who were treated and who were compliant had fewer complications.

There was no sex predilection for incidence of complications.

Out of all the complications, macrosomia and hypoglycaemia had good correlation with maternal glycemic levels.

Asphyxia and birth injuries were more common among macrosomic babies.

Macrosomic babies are usually associated with other complications and hence should be admitted in nursery for close monitoring.

Caesarean deliveries were more common among diabetic mothers.

Hypocalcemia, hyperbilirubinemia and polycythemia in infants born to diabetic mothers were not correlating with maternal blood sugar control as these complications could be caused by various other reasons.

Most of the complications are preventable and hence a meticulous antenatal care is essential.

Early recognition, precise assessment and appropriate management of complications as per guidelines would reduce the mortality and morbidity among babies born to diabetic mothers.

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PROFORMA

Name :

Age / Sex :

Address :

Antenatal Details :

Married since –

Consanguinity –

Age during conception –

Previous IUD / Abortions / Still birth –

Booked and Immunised / Not –

Confirmation of pregnancy –

Gestational Diabetes / Pre gestational Diabetes –

Time of diagnosis –

Treatment details –

Compliance –

Natal Details:

Mode of delivery – Normal labour/ Foreceps/ Vaccum/ Emergency LSCS / Elective LSCS.

Place of delivery.

Liquor – Clear / Meconium stained.

Birth injuries.

Perinatal hypoxia.

Fetal outcome – Abortion/ IUD/ Still born/Term delivery/ Preterm delivery.

Clinical examination of newborn :

AGA / SGA / LGA.

IUGR – Yes / No.

Birth weight.

Head circumference.

Chest circumference.

Oesophageal patency.

Anal patency.

Caput / Cephalhematoma / Subgaleal bleed.

Birth injuries – Fracture humerus / Fracture clavicle / Fracture femur / Erb's palsy/ Klumpkey's palsy.

Seizures – Yes / No.

Respiratory distress – Yes / No.

Cyanosis – Yes / No.

Icterus – Yes / No.

Plethora – Yes / No.

Central Nervous System – Alert / Drowsy.

Laboratory investigations :

In Mother : HbA_{1c} .

In Neonates :

Blood Sugar Values at 1hr, 2hr, 3hr, 6hr, 12hr, 24hr, 36hr and 48hr.

Serum Bilirubin Values – Total, Direct and Indirect.

PCV Values at 1hr and 24 hr.

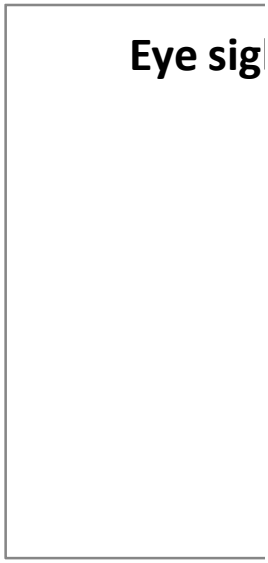
Serum Calcium Values.

Chest Radiography (in babies with respiratory distress).

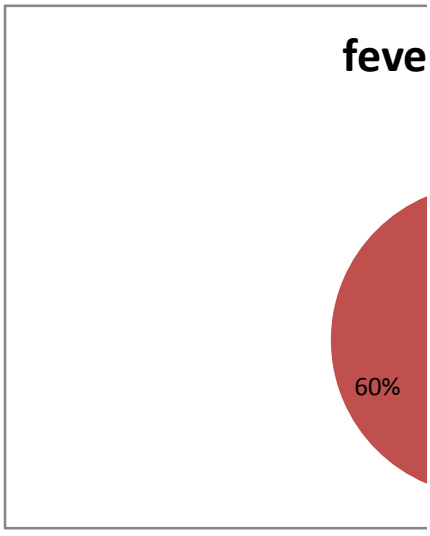
Age	Sex	Delivery	HAB1C	Hypoglycemia	Polycytemia
1	Female	Normal	6	No	No
2	Male	Normal	7	No	No
3	Female	Normal	8	Yes	Yes
4	Male	Normal	6	No	No
5	Female	Normal	7	Yes	Yes
6	Male	Normal	8	No	No

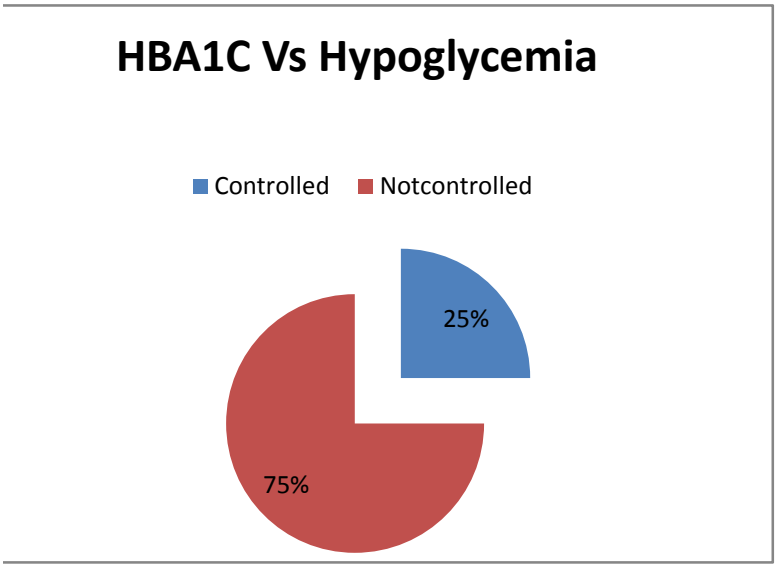
HBA1C	Hypoglycemia
Controlled	25%
Notcontrolled	75%

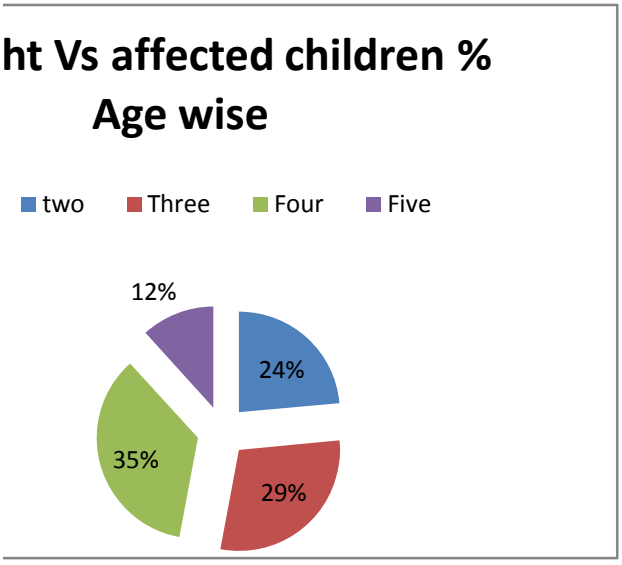
Eye Sight	Affected %
one	15%
two	20%
Three	25%
Four	30%
Five	10%

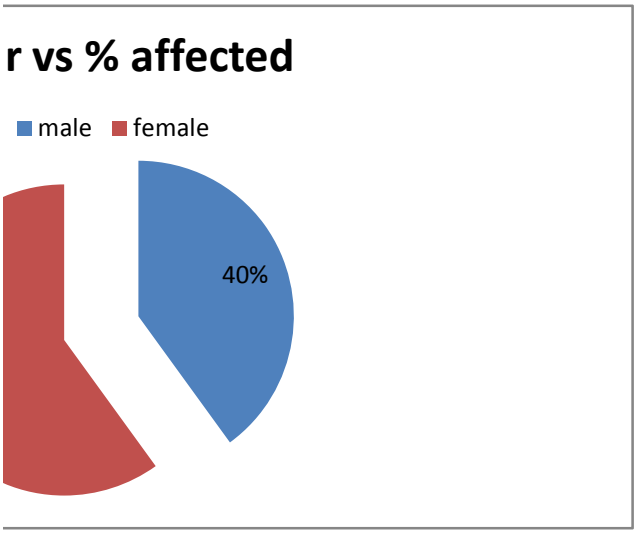


fever	% affected
male	40%
female	60%




HBA1C Vs Hypoglycemia
Controlled
Notcontrolled
25%
75%


ht Vs affected children %
Age wise
two
Three
Four
Five
12%
24%
35%
29%


r vs % affected
male
female
40%

S.NO	AGE	SEX	ADDRESS	BOOKED	GDM/DM	DIAG-AT	TREATMENT	COMPLIANCE	DELIVERY	HbA1C	ASPHYXIA	INJURIES	B.WT	FETAL OUTCOME		M.SOMIA	HYPOGLY	HYPOCAL	HYPERBIL	RDS	POLYCY	1hr	2hr	3hr	6hr	12hr	24hr	36hr	48hr	calcium	PCV
1 .	1/365	Male	Kayathar	Yes	GDM	7th mon	Diabetic diet	Not compliant	LSCS	5.7	No	Nil	3.32	Term	AGA	No	Yes	No	No	No	No	57	32	46	75	94	82	88	90	9.86	52.5
2 .	1/365	Male	Palai	Yes	DM	2yrs	Insulin	Compliant	LSCS	5.25	No	Nil	3.04	Term	AGA	No	Yes	No	Yes	No	No	62	73	56	31	105	98	62	78	10.2	56.23
3 .	1/365	Male	Manur	Yes	GDM	9th mon	Diabetic diet	Compliant	LSCS	5.26	No	Nil	2.61	Term	AGA	No	No	Yes	No	No	No	72	56	105	88	80	75	110	98	6.4	49.06
4 .	1/365	Male	T.Veli	Yes	GDM	9th mon	Insulin	Compliant	LSCS	10.48	No	Nil	4.38	Term	LGA	Yes	Yes	No	Yes	No	No	48	31	27	108	72	85	76	79	9.21	53.78
5 .	1/365	Male	K.Patti	Yes	GDM	8th mon	Insulin	Compliant	LSCS	4.95	No	Nil	2.31	Term	SGA	No	No	No	No	No	No	85	77	112	98	104	152	99	80	9.73	47
6 .	1/365	Female	Sivagiri	No	GDM	7th mon	Insulin	Not compliant	LSCS	7.22	No	Nil	4.04	Term	LGA	Yes	No	No	No	No	No	62	78	103	128	91	109	88	79	10.85	51.69
7 .	1/365	Male	N.Neri	Yes	GDM	7th mon	Diabetic diet	Compliant	LN	5.41	No	Nil	2.11	Preterm	AGA	No	No	No	Yes	Yes	No	78	64	83	62	108	71	128	135	9.86	46.07
8 .	1/365	Male	Kayathar	Yes	GDM	9th mon	Insulin	Compliant	LSCS	5.02	No	Nil	3.71	Term	AGA	No	No	No	No	No	No	82	114	91	70	156	98	120	142	7.91	52.47
9 .	1/365	Female	Ambai	Yes	GDM	7th mon	Not treated	-	LSCS	11.09	No	Nil	4.28	Term	LGA	Yes	Yes	No	No	No	No	34	53	107	65	82	110	98	144	10.79	50.12
10 .	1/365	Female	Tenkasi	Yes	GDM	8th mon	Diabetic diet	Not compliant	LN	7.28	Yes	Yes	4.01	Term	LGA	Yes	Yes	No	Yes	No	No	47	27	35	82	79	92	88	72	7.42	55
11 .	2/365	Male	Pettai	Yes	DM	10yrs	Insulin	Compliant	Foreceps	11.46	Yes	Yes	4.27	Term	LGA	Yes	Yes	Yes	Yes	No	Yes	51	29	22	54	84	75	106	94	5.7	68.25
12 .	1/365	Male	K.Patti	Yes	GDM	9th mon	Insulin	Not compliant	LSCS	8.27	No	Nil	4.18	Term	LGA	Yes	Yes	No	No	No	No	37	78	124	97	115	82	72	127	9.87	48.04
13 .	1/365	Male	Ambai	Yes	DM	5yrs	Insulin	Compliant	LSCS	10.93	Yes	Nil	4.05	Term	LGA	Yes	No	Yes	No	No	No	68	92	133	108	99	113	97	75	6.4	54.7
14 .	1/365	Male	Kadayam	Yes	GDM	7th mon	Diabetic diet	Compliant	LSCS	5.12	No	Nil	2.07	Preterm	AGA	No	No	No	Yes	Yes	Yes	84	71	78	93	98	102	87	73	10.28	70.67
15 .	1/365	Male	Sivagiri	Yes	GDM	7th mon	Insulin	Compliant	LSCS	4.51	No	Nil	3.12	Term	AGA	No	No	No	No	No	No	92	86	118	97	72	126	168	156	8.64	51.45
16 .	1/365	Male	Manur	Yes	GDM	9th mon	Not treated	-	LSCS	10.15	No	Nil	4.14	Term	LGA	Yes	No	No	Yes	No	Yes	84	67	102	126	84	146	97	112	10.32	70.08
17 .	1/365	Female	T.Veli	Yes	GDM	4 Days	Insulin	Compliant	LSCS	7.41	No	Nil	4.28	Term	LGA	Yes	No	No	No	No	No	97	78	127	109	88	132	118	137	8.42	53.73
18 .	1/365	Male	Valliyur	Yes	GDM	7th mon	Diabetic diet	Compliant	Foreceps	5.9	Yes	Yes	4.07	Term	LGA	Yes	Yes	No	Yes	No	No	44	37	86	112	91	108	72	96	9.65	48.12
19 .	1/365	Male	K.Patti	Yes	GDM	9th mon	Diabetic diet	Compliant	LSCS	9.17	No	Nil	4.23	Term	LGA	Yes	Yes	No	No	No	No	49	53	47	25	37	76	105	84	11.56	57.94
20 .	1/365	Male	Tenkasi	Yes	GDM	3 Days	Not treated	-	LSCS	8.21	No	Nil	4.19	Term	LGA	Yes	Yes	No	No	No	No	35	57	92	136	114	157	94	98	9.45	58.34
21 .	1/365	Female	Ambai	Yes	GDM	9th mon	Insulin	Not compliant	LSCS	11.08	No	Nil	4.03	Term	LGA	Yes	Yes	No	No	No	No	52	36	40	76	105	89	92	96	10.42	51.73
22 .	1/365	Male	K.Kurichi	Yes	GDM	7th mon	Insulin	Compliant	LSCS	5.51	No	Nil	3.87	Term	AGA	No	Yes	No	No	No	No	61	56	27	38	86	74	108	87	9.76	62.16
23 .	1/365	Male	Palai	Yes	DM	2 yrs	Insulin	Compliant	LSCS	7.15	Yes	Yes	4.38	Term	LGA	Yes	Yes	No	No	No	No	58	31	64	112	91	146	109	124	8.71	47.98
24 .	1/365	Male	S.vaikun	No	GDM	9th mon	Diabetic diet	Not compliant	LSCS	10.22	No	Nil	4.01	Term	LGA	Yes	No	No	No	No	No	76	104	94	121	86	102	92	98	7.94	54.72
25 .	1/365	Male	Tenkasi	Yes	GDM	7th mon	Diabetic diet	Compliant	LSCS	4.86	No	Nil	3.17	Term	AGA	No	No	No	No	No	No	84	65	107	86	80	92	76	91	10.78	59.41
26 .	1/365	Female	Pettai	Yes	GDM	7th mon	Diabetic diet	Compliant	LSCS	5.69	No	Nil	3.45	Term	AGA	No	No	No	Yes	No	No	74	68	79	127	148	97	110	96	9.28	54.3
27 .	1/365	Male	Ambai	Yes	GDM	7 Days	Insulin	Compliant	LSCS	9.18	No	Nil	4.21	Term	LGA	Yes	Yes	Yes	Yes	No	No	58	32	64	105	86	82	107	118	6.76	57.58
28 .	1/365	Male	V.Puram	Yes	GDM	8th mon	Diabetic diet	Compliant	LSCS	5.04	No	Nil	2.14	Preterm	AGA	No	No	No	No	Yes	No	68	94	75	99	132	101	86	97	9.86	49.71
29 .	1/365	Female	Alangulam	Yes	DM	3 yrs	Insulin	Compliant	LSCS	10.52	No	Nil	4.03	Term	LGA	Yes	No	No	No	No	No	92	65	99	108	86	114	135	121	10.43	55.6
30 .	1/365	Male	Manur	Yes	DM	5 yrs	Insulin	Compliant	LSCS	9.82	No	Nil	1.82	Preterm	AGA	No	No	No	Yes	Yes	No	80	76	105	98	74	97	112	120	8.6	50.37
31 .	1/365	Female	Tenkasi	Yes	GDM	7th mon	Diabetic diet	Compliant	LN	6.56	No	Nil	3.21	Term	AGA	No	No	No	No	No	No	77	96	124	102	97	118	162	125	8.25	52.11
32 .	1/365	Male	K.Patti	Yes	GDM	9th mon	Diabetic diet	Compliant	LSCS	10.29	No	Nil	4.4	Term	LGA	Yes	Yes	No	No	No	No	54	27	33	78	105	86	80	94	10.48	58.44
33 .	1/365	Male	Sivagiri	Yes	GDM	7th mon	Insulin	Compliant	LSCS	5.42	No	Nil	2.75	Term	AGA	No	No	Yes	Yes	No	Yes	58	82	66	114	109	89	132	104	5.24	67.43
34 .	1/365	Male	Kayathar	Yes	GDM	7th mon	Insulin	Compliant	LSCS	4.77	No	Nil	2.23	Term	SGA	No	No	No	No	No	No	84	108	72	96	128	86	112	168	9.74	52.79
35 .	1/365	Male	S.vaikun	Yes	GDM	9th mon	Diabetic diet	Compliant	LN	9.44	Yes	Yes	4.16	Term	LGA	Yes	Yes	No	Yes	No	No	59	76	37	104	152	107	98	134	8.91	50.46
36 .	1/365	Male	Tenkasi	Yes	GDM	4 Days	Insulin	Compliant	LSCS	9.16	No	Nil	4.32	Term	LGA	Yes	Yes	No	No	No	No	47	29	38	86	64	108	87	126	10.21	53.7
37 .	1/365	Female	S.Kulam	Yes	DM	2 yrs	Insulin	Compliant	LSCS	5.68	No	Nil	3.75	Term	AGA	No	No	No	No	No	No	69	92	144	121	97	127	102	96	9.85	57.55
38 .	1/365	Female	Kadayam	Yes	GDM	7th mon	Diabetic diet	Not compliant	LSCS	10.21	No	Nil	4.08	Term	LGA	Yes	No	Yes	No	No	No	96	84	117	132	114	96	148	165	6.1	47
39 .	1/365	Male	R.Palayam	Yes	GDM	7th mon	Insulin	Compliant	LN	4.99	Yes	Nil	2.02	Preterm	SGA	No	No	Yes	Yes	No	No	70	55	94	128	105	99	84	116	5.54	54.13
40 .	1/365	Male	Tenkasi	Yes	DM	3 yrs	Insulin	Compliant	LSCS	11.12	No	Nil	4.27	Term	LGA	Yes	Yes	No	No	No	No	32	41	76	30	108	136	97	125	9.76	56.02
41 .	1/365	Male	Ambai	Yes	GDM	9th mon	Insulin	Not compliant	LSCS	6.07	No	Nil	2.94	Term	AGA	No	No	No	No	No	No	84	76	82	111	86	127	104	137	10.08	57.9
42 .	1/365	Male	S.Malli	No	GDM	9th mon	Diabetic diet	Not compliant	LSCS	8.24	No	Nil	2.61	Term	AGA	No	No	No	No	No	No	76	112	68	97	146	114	123	144	9.36	56.92
43 .	1/365	Female	Panakudi	Yes	GDM	7th mon	Diabetic diet	Compliant	LSCS	5.29	No	Nil	3.8	Term	AGA	No	No	No	No	No	No	81	87	95	62	108	102	84	99	8.45	51.72
44 .	1/365	Male	V.M.Sath	Yes	GDM	7th mon	Insulin	Compliant	LSCS	4.86	No	Nil	1.62	Preterm	AGA	No	No	No	Yes	Yes	No	56	92	78	104	82	79	101	132	11.42	54.1
45 .	1/365	Male	K.kulam	Yes	GDM	5 Days	Insulin	Compliant	LSCS	6.37	No	Nil	4.01	Term	LGA	Yes	Yes	No	No	No	No	52	60	32	38	84	109	87	92	10.96	51.49
46 .	1/365	Male	Tenkasi	Yes	GDM	4 Days	Not treated	-	LSCS	10.46	No	Nil	4.3	Term	LGA	Yes	No	No	No	No	No	74	112	156	98	114	132	167	156	8.4	57.2
47 .	1/365	Male	V.Puram	Yes	GDM	7th mon	Not treated	Compliant	LSCS	9.23	No	Nil	4.24	Term	LGA	Yes	Yes	No	No	No	No	36	42	26	38	75	124	96	88	9.05	52.34
48 .	1/365	Male	Manur	Yes	GDM	7th mon	Diabetic diet	Compliant	LN	5.89	Yes	Yes	4.25	Term	LGA	Yes	Yes	No	Yes	No	No	54	62	35	42	84	76	64	125	8.23	50.4
49 .	1/365	Male	Ambai	Yes	DM	6 yrs	Insulin	Compliant	LSCS	9.42	No	Nil	4.31	Term	LGA	Yes	Yes	No	No	No	No	32	40	76	102	86	148	114	178	10.09	48.76
50 .	1/365	Female	K.Patti	Yes	GDM	8th mon	Insulin	Compliant	LSCS	5.08	No	Nil	2.7	Term	AGA	No	No	No	Yes	No	No	66	52	89	158	96	112	97	90	9.47	52.81
51 .	1/365	Female	Ambai	Yes	GDM	8th mon	Insulin	Compliant	LSCS	5.22	No	Nil	3.74	Term	AGA	No	No	No	No	No	No	70	121	104	98	146	140	105	123	8.54	58
52 .	1/365	Male	Manur	Yes	GDM	8th mon	Diabetic diet	Not compliant	LSCS	9.49	Yes	Nil	4.42	Term	LGA	Yes	Yes	No	No	No	No	32	63	75	108	94	176	132	98	8.9	50.8